The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. 117, No. 24 — COPYRIGHT, 1941, BY AMERICAN MEDICAL ASSOCIATION CHICAGO, ILLINOIS — DECEMBER 13, 1941

THE SUDDEN DEATH OF PATIENTS WITH FEW SYMPTOMS OF HEART DISEASE

GEORGE V. LEROY, M.D.
AND
S. SINCLAIR SNIDER, M.D.
CHICAGO

The sudden death of a patient with disease of the coronary arteries is rarely instantaneous in the sense that the person appears perfectly well one minute and is dead the next. It is instantaneous in the sense that a person with few or many symptoms suddenly ceases to live. The absence of a gradual decline in strength that might indicate a fatal outcome contributes to the unexpectedness of this type of death. In the first few hours or days after the appearance of symptoms that should suggest interference with the coronary blood supply, myocardial insufficiency is seldom severe enough to cause fatal congestive failure, and warning of impending difficulty that is obvious to the casual observer is often lacking. The type of patient we wish to discuss belongs in a strictly limited group: He does not have the dramatically severe syndrome of excruciating thoracic and brachial pain with shock and collapse. Nor does he necessarily show soon after the infarction striking evidence of congestive failure, such as pulmonary edema, orthopnea or failure of the right ventricle. The 10 whose histories we present were selected from a large number of patients observed during the past year by one of us (Dr. Snider) in the course of his routine work as coroner's physician in Cook County. These patients all died suddenly, many in the course of their ordinary activities, unattended by a physician and in circumstances that brought the death to the attention of the coroner. Such clinical information as was available, with the observations at necropsy, is presented briefly in the case histories. The pertinent data are summarized in table 1.

Symptoms which reasonably could be attributed to myocardial infarction were present in all the patients. The duration of symptoms before sudden death varied from three hours to ten days. Six patients had each consulted a physician who had interpreted the symptoms incorrectly and was genuinely astonished when the patient died suddenly. In 3 cases the patient died when a large infarct of the left ventricle ruptured. One patient was straining on a bed pan, 1 was sitting quietly in a chair and 1 was driving his automobile. Their premonitory symptoms were no more striking than the ones experienced by patients in whom the infarct was smaller. It is outside the scope of this report to discuss the phenomenon of the development of huge infarcts with few symptoms, but it is pertinent to bear in mind that extensive myocardial destruction may occur with little obvious evidence of congestive failure. The important issue raised by contemplation of the remaining 7 patients is an adequate explanation of the mechanism of death. Three (patients 8, 9 and 10) had no readily discernible infarction of the myocardium. In them a portion of the muscle was softer than the rest and slightly discolored but no frankly necrotic changes were visible. Invariably one finds more or less recent changes in the coronary vessels that supply the softened area. It is important to emphasize here that the mode of death is no different from that which occurs with the classic syndrome of coronary occlusion during the first few days. In the remaining 4 patients (patients 4, 5, 6 and 7) the infarct discovered at autopsy was small, often no larger than the healed fibrous areas so frequently seen in the myocardium. All 7 patients in whom the ventricle was not ruptured were chosen because the infarct was either small or recent. This was done to emphasize the fact that the death of such patients can scarcely be explained as the result of loss of an important portion of the cardiac musculature.

It must be pointed out that complete obliteration of the artery supplying the infarcted region is not at all necessary. In the patients in whom the ventricle was ruptured, occlusion of a coronary artery by thrombosis was complete. In 4 of the 7 others (patients 5, 6, 9 and 10) occlusion was complete, but in 3 (patients 4, 7 and 8) there was only partial restriction of the vascular channels. Of a recently reported large series of necropsy studies [1] of hearts with myocardial infarction, only 60 per cent showed complete thrombosis of a coronary artery. The rest of the hearts had acquired the infarction as a result of changes in the vicinity of an atherosclerotic plaque or around the coronary ostiums which seriously embarrassed without actually shutting off the coronary blood flow. The changes around the ostiums were usually syphilitic, and those around the plaque were subintimal hemorrhages, ruptures of the plaque or hemorrhages into the area of atheroma.[2]

One may summarize the observations presented and the general attitude of clinicians on sudden death from cardiac disease as follows:

1. Examination of the heart of a victim of sudden death rarely fails to disclose lesions in one or more of

Dr. LeRoy is Searle Research Fellow.

This study was aided by a grant from the Billings Medical Club of Chicago.

From the Department of Medicine, Northwestern Medical School, and St. Luke's Hospital.

Read before the joint meeting of the Section on Practice of Medicine and the Section on Pharmacology and Therapeutics at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 6, 1941.

1. Master, A. M.; Gubner, Richard; Dack, Simon, and Jaffe, H. L.: Differentiation of Acute Coronary Insufficiency with Myocardial Infarction from Coronary Occlusion, Arch. Int. Med. **67**: 645-657 (March) 1941.

2. Horn, Henry, and Finkelstein, L. E.: Arteriosclerosis of the Coronary Arteries and the Mechanism of Their Occlusion, Am. Heart J. **19**: 655-682 (June) 1940.

the coronary arteries which impede or obstruct the blood flow. In location and extent these lesions often cannot be distinguished from similar ones in patients dead from other causes.

2. The myocardium of a victim of sudden cardiac death nearly always contains an infarct. In the relatively few instances in which there are no symptoms antecedent to death, neither a softening nor a clearly demarcated infarct may be found in association with the vascular lesion. In all patients who have had symptoms —however mild—for a few hours or more before death, the myocardium is damaged. The infarct if early is simply a softened darker red area with some dilatation of the arterioles in the overlying epicardial fat; or there may be a discrete region of muscular necrosis with hemorrhagic discoloration and fatty changes. The infarct may be of any size and in any position; in this respect it differs in no way from the fibrous scar of a healed infarct that is seen so frequently.

In an effort to explain the paradox that presumably identical cardiac vascular and muscular lesions may or may not cause sudden death, most writers have assumed the intervention of some sort of a reflex disturbance of cardiac function severe and sudden enough to prove fatal. Ventricular asystole and ventricular fibrillation [3] have been demonstrated as the terminal mechanism in the few instances in which electrocardiograms were being made when death occurred. The majority of patients died with ventricular fibrillation, but the mode of its development has not been clearly understood. It is obvious that insight into the causation of this fatal arrhythmia should provide clues to aid one in combating its onset. The greatest difficulty in reconstructing the series of events responsible for a sudden death is that this must be done in retrospect, with only dead tissues as building materials. Other difficulties are the variability of the vascular and muscular lesions and—as we have indicated—the fact that similar lesions may not cause death. Finally, it is apparent that age, physiologic condition and inherent biologic differences must be important determining factors. Animal experimentation eliminates fairly well most of the confusing factors except the inherent ones. We think these are the most important.

The effect of the ligation of a coronary artery of a dog has been studied by many workers. The number of animals that survived after ligature of the circumflex branch of the left coronary artery varied considerably. In table 2 are collected some of the earlier data. In 1939 Hall and his colleagues [4] reported the first of a great many experiments in which ligation was performed on conscious animals through the tightening of a previously applied loose ligature. In general these workers were studying alterations in mortality that followed various disturbances of the innervation of the heart. When one of us (Dr. LeRoy) in 1940 commenced a study of this problem, an attempt was made to prepare anesthetized animals which would have the same mortality as conscious ones. The considerations behind this step were not entirely humanitarian. It was thought, however, that the subjective sensation of pain evidenced by the conscious dog might reinforce an existing reflex or even initiate one. The fact that use of anesthesia with morphine and pentobarbital sodium resulted in the same mortality as that for conscious dogs seems to indicate that the sensation of pain is a by-product rather than an essential feature of the mechanism of sudden death. This phase of our work was prompted by study of patients of the type referred to earlier, in whom pain was a minor element in the series of events that followed occlusion of a coronary artery.

In our laboratory a strictly standardized procedure is followed for the production of acute coronary occlusion. After the induction of morphine and pentobarbital sodium anesthesia (aseptic precautions being observed) in a dog a ligature is placed on the circumflex branch of the left coronary artery. Subsequently the heart may be left exposed for direct observation and color photography.[5] In other dogs the thorax was closed, and the effect of certain drugs on survival was studied. With a large number of such preparations the mortality after coronary occlusion was 75 per cent. The value is the same as that found by Hall and by us for conscious animals. In the discussion of our results, and

TABLE 1.—*Summary of Case Histories*

Patient	Age	Sex	Symptoms: Duration	Symptoms: Type	Autopsy: Myocardium	Autopsy: Arteries *	Medical Care
1	50	♂	7 days	Pain in chest, dyspnea	Rupture, infarct, left ventricle	Thrombosis, L.A.D.	No
2	56	♂	3 days	Cold in chest, cough	Rupture, infarct, left ventricle	Thrombosis, L.A.D.	Yes
3	60	♀	10 days	Substernal pain, cough	Rupture, infarct, left ventricle	Thrombosis, L.A.D.	Yes
4	45	♂	16 hours	Fatigue, tightness in chest	Infarct, 2 × 3 cm., apex and septum	Subintimal hematoma, L.A.D.	No
5	41	♂	4 days	Dyspnea, pain in arm	Infarct, 2 × 2 cm., apex, left ventricle	Thrombosis, R.C.A.	Yes
6	39	♂	24 hours	Pain in arms	Infarct, 4 × 5 cm., left ventricle	Thrombosis, L.C.A.	Yes
7	32	♀	4 hours	Nausea, dyspnea, fear	Infarct, 6 × 7 cm., left ventricle	Stenosis, coronary ostiums	Yes
8	49	♂	10 days	Angina pectoris	Softening of apex	Diffuse sclerosis	No
9	41	♂	3 hours	Nausea, substernal pain	Softening, posterior wall of left ventricle	Thrombosis, both circ.	Yes
10	43	♂	4 hours	Nausea, vomiting	Softening of base and septum	Subintimal hematoma, A.B.R.C.A.	No

* L.A.D., anterior descending branch, left coronary artery; R.C.A., right coronary artery; L.C.A., circumflex branch, left coronary artery; A.B.R.C.A., aberrent branches, right coronary artery; both. circ., circumflex branches, right and left coronary arteries.

3. Thompson, Ivan: Ventricular Fibrillation Causing Sudden Death, J. A. M. A. **116**: 2583-2585 (June 7) 1941. Goodrich, Ben E., and Needles, R. J.: Terminal Cardiac Mechanism in Coronary Artery Disease, Am. Heart J. **20**: 637-640 (Nov.) 1940. Weiss, Soma: Instantaneous "Physiologic" Death, New England M. J. **223**: 793-797 (Nov. 14) 1940.

4. Manning, G. W.; McEachern, C. G., and Hall, G. E.: Reflex Coronary Artery Spasm Following Sudden Occlusion of Other Coronary Branches, Arch. Int. Med. **64**: 661-674 (Oct.) 1939. McEachern, C. G.; Manning, G. W., and Hall, G. E.: Sudden Occlusion of Coronary Arteries Following Removal of Cardiosensory Pathway, ibid. **65**: 661-670 (April) 1940.

5. When this paper was presented at the Annual Session a color film was shown.

in our comments on Hall's work, the phrase sudden death will be applied to animals which died within twenty-four hours after ligation of the circumflex branch. All mortality referred to concerns only cases of sudden death.

The sequence of events after the occlusion of the circumflex branch of the left coronary artery is as follows:

1. At the instant of occlusion the blood pressure falls suddenly, and a shower of extrasystoles or well defined bradycardia appears during an interval of about fifteen seconds. The electrocardiogram is usually unchanged for at least thirty seconds. The conscious dog may or may not experience pain.

2. One minute after occlusion the blood pressure may be lower or normal. A well demarcated zone of ischemia is present in the part of the heart deprived of its blood supply. Typical electrocardiographic changes are occurring: elevation of the RS-T segment in all leads, with exceptionally high elevation in leads 2 and 3. The pulse is generally more rapid, and a variable number of extrasystoles occur.

3. Five minutes after occlusion the blood pressure is usually falling; the pulse is more rapid, and showers of extrasystoles and short runs of ventricular tachycardia are seen in most dogs. The myocardial infarct is deeper in color, and the ischemic myocardium bulges outward during systole. The electrocardiographic changes are more prominent. In the conscious dog there is usually evidence of more or less pain, altered respirations and struggling.

4. At any time after five minutes one can see little further alteration in the situation. The infarct may become somewhat darker, and the arrhythmias are more frequent and more prolonged. In the animal destined to die suddenly the only difference one can see is a greater amount and variety of arrhythmias.

5. Suddenly the entire uninfarcted part of the myocardium is seen to swell; dilation of the heart is occurring. The uninvolved part of the myocardium at the same time becomes darker until the margin of the infarct disappears. The contractions of the ventricle become feeble, rapid and irregular, and paroxysmal ventricular tachycardia is recorded electrocardiographically. Suddenly a portion of the uninfarcted area of the myocardium begins to fibrillate, and in a few seconds the process spreads until generalized ventricular fibrillation is established. The blood pressure has then fallen to zero, and the dog, after giving a convulsive gasp, is dead.

This description is a synthesis of about fifty observations made on conscious animals and on suitably anesthetized ones with the heart exposed. Colored motion pictures of many preparations were taken and studied carefully. In every respect our description of the sudden death of a dog with experimental coronary occlusion agrees with that published by the Toronto workers. One feature that we have seen is that when death occurs rapidly—that is in less than thirty minutes—no infarct is visible afterward, whereas if the dog survives longer a distinctly darker infarct is found.

TABLE 2.—*Mortality After Experimental Occlusion of Circumflex Branch of Left Coronary Artery*

Investigator	Mortality, Percentage
Cohnheim and von Schulthess-Rechberg	100
Porter	64
Smith	57
Clerc and associates	100
Parade	100

* See also Burchell, H. B.: Adjustments in Coronary Circulation After Experimental Coronary Occlusion, Arch. Int. Med. 65:240-262 (Feb.) 1940.

The main feature of the experiments is the clear demonstration that there is a generalized coronary vasoconstriction subsequent to the myocardial infarction. This is clearly seen in the animal (and is shown in the colored pictures) when the uninfarcted part of the myocardium gradually becomes as dark as the infarct just before ventricular fibrillation commences. The existence of such a coronary vasoconstriction has been demonstrated by us in other experiments, in which the blood flow in one coronary artery was measured with a thermostromuhr while another artery was ligated. It seems apparent to us that the ischemia of the myocardium is provoked by a reflex coronary vasoconstriction and that it is the factor that causes ventricular fibrillation in the animal whose myocardium is susceptible to the development of this arrhythmia. The main pathways of the reflex arc may be inferred from existing data. Many workers have shown that painful afferent impulses from a myocardial infarct travel centrally through the cardiosensory nerves, the stellate ganglions and the upper five or six thoracic sympathetic ganglions. These workers have shown also that cardiosensory denervation prior to infarction reduces the mortality strikingly. The efferent impulses provoking vasoconstriction must travel from the brain to the coronary arteries in the vagus nerves, since they are the only generally accepted conductors of such activity.

TABLE 3.—*Mortality After Ligation of Circumflex Branch of Left Coronary Artery*

Experimental Conditions		Incidence of Sudden Death, Percentage	Investigator *
Anesthetic	Modifying Circumstances		
None	Both stellate ganglions and upper five thoracic sympathetic ganglions removed	10	H
None	One stellate ganglion and upper five thoracic sympathetic ganglions on same side removed	33	H
Ether (deep anesthesia used)	None	25	H
Morphine-pentobarbital sodium	Xanthine drugs administered after ligature	25	L
Morphine-pentobarbital sodium	Atropine sulfate administered	35	L
None	Atropine sulfate and xanthine	30	L
None	Quinidine sulfate administered	55	H
None	Papaverine hydrochloride administered	50	H
None	None	75	H
Morphine-pentobarbital sodium	None	75	L
None	None	75	L

* H, Hall, of Manning, McEachern and Hall;[4] L, LeRoy.

Many experiments were performed in Hall's laboratory and in ours in an attempt to modify the incidence of sudden death after experimental coronary artery occlusion. In table 3 are collected the data. The greatest reduction in mortality occurs when the afferent portion of the lethal reflex arc is interrupted surgically: Bilateral removal of the stellate ganglions and the upper five thoracic sympathetic ganglions reduced the mortality in conscious dogs from a control value of 75 per cent to only 10 per cent. Unilateral sympathectomy reduced the mortality to only 33 per cent. Surgical ether anesthesia sufficiently depresses all reflex activity, so that ligations in which it was used resulted in a mortality of only 25 per cent. On the other hand, light anesthesia with morphine and pentobarbital sodium had so little influence on the reflex in question that the mortality was the same as for conscious dogs, 75 per cent. Administration of atropine sulfate in appropriate

doses reduced the mortality to about 35 per cent. A death rate of 25 per cent was observed after the administration of active coronary vasodilating drugs, the xanthine derivatives theobromine and theophylline. When atropine sulfate and theophylline with ethylenediamine were used, the death rate, in conscious dogs, was 30 per cent. Use of quinidine sulfate, which has no action on either the coronary vessels or the reflex arc, reduced the mortality to only 55 per cent.

From the experiments described one can safely conclude that sudden death after ligation of a coronary artery is due to ventricular fibrillation.[6] This arrhythmia is caused by generalized myocardial ischemia, which is accomplished by active vagal coronary vasoconstriction. This unfortunate stimulation of the vagus is produced reflexly by afferent impulses arising in the myocardial infarct and transmitted centrally through the sympathetic nerves. Amelioration of the vagal vasoconstriction, whether by cardiosensory denervation, by the use of atropine, by deep surgical anesthesia or by active coronary vasodilatation secured with the xanthine drugs, reduces the incidence of sudden death appreciably. The extent to which the reflex operates in the absence of efforts to interfere with it depends on certain inherent biologic phenomena: the stimulation threshold of the cardiosensory sympathetic nerves, the tone of the vagus and the susceptibility of the uninfarcted part of the myocardium to the influence of ischemia. Certainly these factors are difficult if not impossible to evaluate.

We think it is perfectly reasonable to apply to human beings the information derived from our studies on dogs. In each one observes the same unpredictable outcome from apparently similar lesions. In each sudden death is obviously not due to simple failure of the contractile power of the myocardium in the infarct alone. In most cases in which sudden death of a patient with recent coronary occlusion has been studied electrocardiographically, the same mechanism—ventricular fibrillation—has occurred that we have seen in the dog. Therefore it is reasonable to employ the means discovered by experimentation on the dog in an attempt to reduce the mortality among patients.

We think the data we have presented are important for two reasons. First, the realization that reflex phenomena are responsible for the sudden death of a patient with myocardial infarction is valuable. Appreciating this, one should be less prone to pass over lightly mild symptoms or mild physical signs that suggest coronary artery disease. Bearing in mind that these mild symptoms may be the sole evidence of an infarct that can at any moment initiate fatal ventricular fibrillation, one will be less likely to ignore them. The sudden onset in a previously healthy person of substernal oppression, mild pain, symptoms of indigestion or brachial pain or discomfort unrelated to injury should arouse suspicion. Suspicion should be doubled if the symptoms are aggravated by exertion, especially if they are associated with dyspnea. Dyspnea and fatigue occurring suddenly and experienced under conditions under which it is not usually felt should always excite suspicion. With suspicion aroused, the patient should be examined with great care. We have little doubt that most patients with mild symptoms of myocardial infarction have some discoverable cardiac abnormality, such as showers of extrasystoles, pulsus alternans, perhaps a friction rub and a blood pressure lower than the usual reading. If facilities are available an electrocardiogram should be made, and small deviations from normal—particularly in the RS-T segment—should be given serious consideration. The most important point clinically is that suspicion rather than certainty should govern one's conduct.

The second reason why we attach significance to the facts presented is that an answer is supplied to the question of the physician who says "All right, this patient may have a myocardial infarct; but what can be done about it while the symptoms and signs are so indefinite except to wait and see and to meet whatever situations arise?" The fault in this attitude, of course, is that the first serious change in the patient's status may be the onset of fatal ventricular fibrillation! The animal studies offer some indications of useful, reasonable therapeutic measures, and from them we have learned that certain agents are available which will minimize or counteract the lethal reflex. Rest alone for the patient suspected of having a myocardial infarct is undoubtedly valuable, because it decreases the demand of the myocardium for blood and thus lessens the effect of any coronary vasoconstriction that might occur; but rest is not enough. Atropine and the coronary vasodilator drugs, such as the salts of theobromine and theophylline, can be given in large amounts by several routes and with no risk. The doses of these drugs that we have used for dogs are approximately equivalent to the doses for human beings. Of atropine sulfate we give dogs about one half the atropinizing dose, or 0.1 mg. per kilogram of body weight. As to patients, one-half the atropinizing dose is about 0.8 mg. (1/75 grain), given intravenously, for the usual adult. Of aminophylline (theophylline with ethylenediamine), for example, we give patients and dogs an initial dose of 5 to 10 mg. per kilogram of body weight intravenously. The usual amount for an adult is 0.48 Gm. in 20 cc. of solution, given intravenously and slowly. The injection may be repeated every four or six hours as the situation suggests. Surely in case of doubt one can do no harm by giving such amounts of atropine and a xanthine derivative while one waits to see what will happen or while one waits for an electrocardiogram to be made. Naturally not all patients who are so treated will survive; but if the same ratio observed in dogs should hold for human beings 1 of 3 will die, instead of 3 of 4. The reduction in mortality is certainly a worth while advantage. It is hard indeed for us to escape the conclusion that many victims of heart attacks whom the coroner's physician sees would not have died had atropine and one of the xanthine derivatives been given and complete bed rest insisted on soon after the onset of the symptoms.

CONCLUSIONS

1. The myocardial infarct responsible for sudden death need not be so large that muscular failure alone is responsible.

2. Myocardial infarction may occur without complete closure of a coronary artery and without the classic syndrome of pain, shock and collapse.

3. A myocardial infarct usually causes some symptoms, even though they may be mild.

4. The sudden death of a patient with infarction of the myocardium is due to a reflex coronary vasoconstriction whose stimulus is the infarct, whose afferent pathway is the cardiosensory innervation and whose efferent pathway is the vagus. The result of this reflex vasoconstriction in a susceptible person is fatal ventricular fibrillation.

6. In our experiments a few instances of partial auricular-ventricular block were seen, but in no case in which death was observed was this the terminal mechanism. The experience of Hall and his associates[4] was similar.

5. The probability of the establishment of this lethal reflex may be decreased by use of certain drugs (atropine and the xanthine derivatives), by cardiosensory denervation and reasonably but to a lesser extent by simple rest in bed.

REPORT OF CASES

CASE 1.—A man aged 50 had complained for about one week of slight but increasing substernal pain. Three days before death increasing dyspnea, orthopnea and more severe pain occurred. He was up and around during the entire time but had received no medical care.

Necropsy disclosed moderate hemopericardium, the origin of which was a small perforation of an aneurysmal dilatation of an infarct of the lateral wall of the left ventricle. Complete occlusion by a thrombus of the descending branch of the left coronary artery was found.

CASE 2.—A man aged 56 ran into a tree while driving home from work. He was dead when passersby reached the wreck. Three days previously he had consulted a physician because of a "cold in the chest" and had received some sort of medication.

Necropsy disclosed moderate hemopericardium, the origin of which was a large rupture of an infarct of the posterior surface of the left ventricle. A complete occlusion of the more distal portion of the circumflex branch of the left coronary artery by a thrombus was observed.

CASE 3.—A woman aged 60 was bedridden, convalescing from a seven week old fracture of the femur. Ten days before death she complained of substernal tightness to her physician, who visited her regularly. This was diagnosed and treated as a "flu-like cold in the chest." Three days before death she had increasingly severe substernal pain, coughing and dyspnea. She died suddenly while getting off a bedpan.

Necropsy disclosed moderate hemopericardium, the origin of which was a large rupture of an infarct at the apex of the left ventricle. A complete occlusion by thrombosis of the anterior descending branch of the left coronary artery was observed. This had formed on a calcified atherosclerotic plaque 1 cm. below the origin of the vessel.

CASE 4.—A man aged 45, a pharmacist, had had a headache all day and had felt tired. About an hour before death he experienced a constant tightness in his chest. He fell over suddenly behind a counter, dead.

Necropsy disclosed a 2 by 3 cm. infarct at the apex of the left ventricle. The anterior descending branch of the left coronary artery was incompletely occluded by an atheromatous plaque, elevated by a fresh subintimal hemorrhage. The left coronary artery was small in caliber, less than one-half the usual size. The posterior wall of the left ventricle was supplied almost entirely by a large branch of the right coronary artery.

CASE 5.—A man aged 41, a linesman, died suddenly at work on a pole. His widow stated that for the preceding four days he had complained of breathlessness and of "neuralgia" in his left arm, aggravated by exertion. He had seen a physician on the morning of the day of his death and had received some pills.

Necropsy disclosed a small infarct, about 2 by 3 cm. in size, with beginning fatty necrotic changes at the apex of the heart involving the septum. A small thrombus attached to an ulcerated atherosclerotic plaque partly occluded a posterior descending branch of the right coronary artery.

CASE 6.—A man aged 39, a milkman, experienced moderately severe pain across the chest and in both arms the day before he died. Despite this pain he was able to finish his deliveries. On his way home he saw a physician, who gave him some pills. He slept poorly but went to work the next day. He was found dead on a prairie a short distance from the start of his milk route.

Necropsy disclosed a 3 by 4 cm. infarct on the lateral wall of the left ventricle. The circumflex branch of the left coronary artery was completely occluded by a thrombus fixed to an ulcerated atheroma.

CASE 7.—A woman aged 32 became nauseated while sitting quietly at home. With the gastrointestinal disturbance there was some substernal tightness and a sensation of fear. A physician was called who diagnosed "intestinal flu" and prescribed some medicine. She lay down to rest and was found dead four hours after the onset of symptoms.

Necropsy disclosed a large infarct of the posterior, apical and septal regions of the left ventricle. The ostium of the left coronary artery was reduced to a diameter of 1.5 mm. by an encircling gumma. The ostium of the right coronary artery was completely obliterated by a recent subintimal hemorrhage in the syphilitic infiltration of this region. Large gummatous pads, some with recent subintimal hemorrhages, encircled the root of the aorta and separated the attachments of the valves.

CASE 8.—A man aged 49, a bricklayer, died suddenly at work. During the preceding week or ten days he had complained of moderate precordial pain on effort. About a year earlier he had suffered from what was diagnosed as coronary occlusion, but the electrocardiogram was asserted to have been "negative."

Necropsy disclosed a slight softening of the apical region, with no distinct infarction. The small blood vessels in the epicardial fat of this region were widely dilated. The anterior descending and the circumflex branches of the left coronary artery were completely occluded by old calcific lesions. There was a diffuse fibrous scar in the posterior wall of the left ventricle. The right coronary artery was smaller than usual, and anomalies of both arteries were seen.

CASE 9.—A man aged 41, a salesman, became sick at work, with gastrointestinal disturbances, substernal pain and little or no evidence of shock. He was taken to a hospital, where his symptoms decreased after morphine was given. He suddenly collapsed and despite efforts at resuscitation died suddenly three hours after the onset of symptoms.

Necropsy disclosed a recent barely discernible infarct of the posterior wall of the left ventricle and of the septum. This was apparently due to occlusion by fresh thrombi attached to atherosclerotic plaques in the circumflex branches of both coronary arteries. There was an old fibrous scar, 3 by 4 cm. in size, in the anterolateral wall of the left ventricle. This was in relation to an old, fibrous, complete occlusion of the anterior descending artery.

CASE 10.—A man aged 43 died suddenly while changing his clothes before starting to work in a factory. He had complained that discomfort in the upper part of the abdomen had awakened him, and he had eaten no breakfast. He had a flannel binder pinned about his abdomen.

Necropsy disclosed a faintly defined infarct around the base of both ventricles and in the region of the septum adjacent. The discoloration extended into the interauricular and the interventricular septums. No septal branch of the left coronary artery could be found. There were two tiny aberrant ostiums in the anterior sinus of Valsalva in addition to the ostium of the right coronary artery. Vessels from the two small ostiums, almost completely occluded by fresh subintimal hemorrhages, were traced to the infarct.

1713 West Ninety-Fifth Street.

ABSTRACT OF DISCUSSION

DR. G. K. FENN, Chicago: The paper of Drs. LeRoy and Snider has attracted my attention for two reasons. First, I have been greatly interested in reflex effects on the coronary circulation. I suspect that such effects may be initiated in many places, both in the heart and from extracardiac sources. Two years ago, before this section and on the same program with Dr. Hall, Dr. de Takats and I submitted evidence which led us to believe that reflex coronary constriction accompanied pulmonary embolism. Last year Drs. Gilbert, LeRoy and I tried to convince this section that we had demonstrated the occurrence of such reflexes arising in the gastrointestinal tract. Now, Drs. LeRoy and Snider have built up a rather clearcut case to show that patients die following unsuspected interference with the coronary circulation and that many of these deaths are not due to the infarct per se or to congestive heart failure. They have integrated these occurrences into experimental work

which seems to indicate quite clearly that many of these deaths are due to reflex coronary constriction with its attendant phenomena, notably ventricular fibrillation. I do not see how we may disregard any longer the importance of these reflex effects. I am interested secondly because of the altered therapeutic approach to the problem that has grown out of all this work. Not long ago morphine was the only drug that received much attention in the treatment of acute coronary occlusion. Now morphine does not overcome the reflex constriction. Indeed, it has been accused of accentuating such effects. Certainly morphine did nothing toward saving the life of the experimental animals. It seems clear therefore that, when morphine must be used, and it frequently must be or is used, something should be added to offset its vagal action. Dr. LeRoy has pointed out the advantages derived from atropine and drugs of the xanthine group. Incidentally, it is surprising how much less morphine is necessary in the management of acute coronary occlusion when the reflex is overcome by the use of atropine and theophylline plus the inhalation of oxygen. I do not believe that the importance of these reflex effects is sufficiently appreciated by the medical profession in general. No harm can come from the application of these remedies to patients for whom the diagnosis is uncertain. The great harm arises in not considering the possibility of an interference with the coronary flow.

DR. HAROLD E. B. PARDEE, New York: There are two parts to this paper, both of importance. The first part calls our attention to the frequency of sudden death of patients who have had only minor symptoms referable to the heart. The preliminary symptoms are due to coronary insufficiency, and we must learn to recognize such symptoms as possibly due to serious coronary occlusion. In these patients we must particularly look for three important diagnostic features: arrhythmia, fall of blood pressure and electrocardiographic changes. Pulsus alternans and pericardial friction are also of importance but are unfortunately rare. We should make a special effort to obtain the electrocardiogram in these cases because the record will often give an indication of myocardial abnormality in cases that are otherwise negative. It is important to emphasize in this connection that, when the limb leads are normal, if we take two precordial leads, one from the parasternal position and one just beyond the apex, we shall obtain positive findings in 12 or 13 per cent of additional cases. The second part of the paper, on the experimental work, confirms the importance of a reflex mechanism in producing the sudden death which occurs not only among patients who have had only slight symptoms due to coronary insufficiency but also among those who have had cardiac infarction with severe symptoms. Certain therapeutic recommendations are based on this experimental work. It is most important to give an adequate dose of atropine in these cases. The initial dose should be at least 1/75 grain (1 mg.) or, better, 1/50 grain (1.3 mg.) and the effect may be continued by 1/150 grain (0.4 mg.) every four or six hours. An adequate dose of the xanthines is also important. We should give from 5 to 7½ grains (0.3 to 0.5 Gm.) by mouth or the same dose intravenously, provided it is given slowly. Doses of this size are necessary to produce the definite therapeutic effects that are needed in such cases. I feel that the correlation of clinical observation and experimental work in this paper is an ideal of what medical research should be and that the therapeutic conclusions Dr. LeRoy has drawn from this work deserve strong consideration.

DR. GEORGE V. LEROY, Chicago: I was pleased that Dr. Fenn brought up the question of the role of morphine, because it is a complicated problem that we have not completely worked through, using the technic we have described here. It should be observed, though, that to get the same mortality in dogs anesthetized with pentobarbital sodium as occurred in conscious dogs it was necessary to add a definite amount of morphine. If the dogs received pentobarbital sodium alone the mortality was between 30 and 40 per cent, about half way between that of dogs under surgical ether and conscious dogs. Morphine definitely made the dogs more liable to die and was used deliberately in preparing these animals. The other thing that I think bears repeated emphasis is the fact that myocardial failure, as seen by the classic signs of edema, cyanosis and dyspnea, is a rather minor feature during the first few days in the majority of patients with myocardial infarctions, especially the patients we talked about with small infarcts. Certainly, in the autopsies of these patients, pulmonary edema and hepatic congestion were the exception rather than the rule. I was pleased to have Dr. Pardee recommend the larger doses of drugs. It is our custom clinically, in the use of atropine to give an additional dose of from 1/50 to 1/75 grain intravenously. The patient is then maintained in atropinization by 1/100 to 1/50 grain at appropriate intervals. The doses of the xanthines in our practice are about 7½ grains every four to six hours in recent occlusions, which parallels very closely milligrams on a kilogram basis, the amounts we used to achieve the results in the dogs.

SIMILARITIES AND DISTINCTIONS BETWEEN SHOCK AND THE EFFECTS OF HEMORRHAGE

VIRGIL H. MOON, M.D.
DAVID R. MORGAN, M.D.
MARSHALL M. LIEBER, M.D.
AND
DONALD McGREW, M.D.
PHILADELPHIA

Clinical similarities between shock and the effects of hemorrhage are so numerous that some authorities have regarded the two as identical. It is believed by many that hemorrhage will produce the complete syndrome of shock—a belief which we formerly supported.[1] We supposed that anoxia in the tissues resulting from the effects of hemorrhage would cause endothelial permeability, hemoconcentration and other features characteristic of shock. Blalock[2] had reported such results, and these coincide with our belief as published; but attempts to substantiate that belief experimentally produced results which were not anticipated.

We made the simple experiment of withdrawing measured amounts of blood from the femoral veins of an unanesthetized dog (no. 338) weighing 10 Kg., allowing intervals of time for physiologic readjustment of the circulation. Erythrocyte counts and hemoglobin determinations were made (by the photoelectric method) before and at intervals during the experiment. Examinations of the blood, including one of the last sample drawn, revealed progressive dilution.

Successive repetitions of the experiment, greater care being taken to approach the death point gradually, gave similar results. Eight dogs were used in unsuccessful attempts to demonstrate a terminal hemoconcentration as a result of simple hemorrhages. The data are summarized in table 1.

It may be seen that dogs vary in their sensitivity to loss of blood. The loss necessary to cause death within twelve to sixty-seven hours ranged from 5.58 to 9.1 per cent of the body weight, but no relation is apparent between this variation and the duration of the experiments. Postmortem examinations were made immedi-

From the Department of Pathology, Jefferson Medical College of Philadelphia.

Because of lack of space, this article has been abbreviated for publication in THE JOURNAL. The complete article appears in the authors' reprints.

The experimental work reported here was supported in part by a donation by Parke, Davis & Co. and in part by a grant from the National Research Council.

Read before the joint meeting of the Section on Surgery, General and Abdominal, and the Section on Orthopedic Surgery at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 6, 1941.

1. Moon, V. H.: Shock and Related Capillary Phenomena, New York, Oxford University Press, 1938, p. 171.

2. Blalock, Alfred: Acute Circulatory Failure as Exemplified by Shock and Hemorrhage, Surg., Gynec. & Obst. **58**: 551 (March) 1934; Shock: Further Studies with Particular Reference to the Effects of Hemorrhage, Arch. Surg. **29**: 837 (Nov.) 1934.

ately, and the visceral appearances in each instance were those characteristic of hemorrhage. The tissues were ischemic, pale and dry; there was no engorgement of the minute vessels, edema, effusions or petechiae. No parenchymal necroses were found by microscopic examination in any of the tissues. Data on these visceral changes were substantiated in all the subsequent experiments on hemorrhage.

In another series the dogs were anesthetized by an intravenous or an intraperitoneal injection of 0.035 Gm. of amytal per kilogram of body weight in order that a continuous record of the arterial blood pressure after hemorrhage might be made. The dogs were bled in measured amounts at intervals from the femoral artery or vein. The duration of the experiment, from the first bleeding to the death of the animal, ranged from four to thirty-one hours. In the experiments of long duration, several bleedings were done on the preceding day, with the dogs in a normal state. Anesthesia and blood pressure readings were initiated only in the last four to six hours of the experiment. The maximal period of anesthesia in the group was six and five-tenths hours. Data procured under these conditions are shown in table 2.

In all instances the viscera were pale, dry and ischemic; no edema, congestion or petechial hemorrhages were found in any tissue at necropsy. The with-

TABLE 1.—*Effects of Hemorrhages in Unanesthetized Dogs*

Dog	Weight *	Blood Drawn, Percentage	Time, Hours	Hemodilution, Percentage
338	10.0	5.9	26	60
339	7.25	8.0	67	81
342	11.3	6.63	23.5	50
343	15.4	5.58	12.5	62
344	12.7	8.14	54	57
345	10.5	7.50	28.5	50
346	10.0	9.1	31.5	77
347	7.9	7.78	32.5	76

* In this and the following tables weights are shown in kilograms and the amount of blood drawn is shown as percentage of the body weight.

drawal of a smaller total volume of blood produced death, and the hemodilution was much less than in unanesthetized dogs. This is exemplified in a comparison of dogs 346 and 373 (table 3).

It may be seen from the comparison that a loss of blood amounting to 9.1 per cent of the body weight caused 77 per cent hemodilution and death in thirty-one and one half hours without anesthesia, while half as great a loss of blood caused death in thirty-one hours the last six and one half of which the dog was under amytal anesthesia. The hemodilution was less than half that shown by the unanesthetized dog. The reactions of anesthetized animals were significantly different from those of unanesthetized ones. In 2 instances, after hemorrhages totaling 2.6 per cent and 2.2 per cent of the body weight respectively the blood remained at approximately the same concentration as before bleeding, and in 2 others a slight temporary rise followed by a decline in the concentration occurred.

Significant differences were noted between the behavior of dogs after hemorrhages and that of dogs in a state of shock from the absorption of tissue substance. The former were active and did not appear ill; there was no vomiting, diarrhea or evidence of urinary disturbance. Dogs in shock uncomplicated by narcosis are inactive and manifest each of the features of illness mentioned. These apparent contrasts led us to make further comparisons, both of observations recorded by other investigators and of features developing under experimental conditions. Surprisingly few data are on record concerning physiologic disturbances associated with either shock or hemorrhage, and no detailed comparison of these has been found.

TABLE 2.—*Effects of Hemorrhage in Dogs Under Amytal Anesthesia*

Dog	Weight	Blood Drawn, Percentage	Time, Hours	Hemodilution, Percentage
352	21.8	6.2	4.0	35
354	13.0	3.15	4.5	14 *
358	11.8	2.61	6.5	11
360	11.3	2.2	6.5	0 *
364	8.2	3.9	24.5	48
365	8.0	3.75	28	60
366	6.7	3.58	24.5	46
367	11.4	5.0	26	40
368	11.8	5.3	25.5	43
369	11.3	5.3	25.5	53
372	11.2	4.74	6	34
373	10.8	4.48	31	32 *
375	8.6	3.14	25	55

* Dog 354, temporary hemoconcentration followed by dilution (see text); dog 360, neither concentration nor dilution; dog 373, slight concentration followed by dilution.

SUSCEPTIBILITY TO HEMORRHAGE

A comparison was made between the response to hemorrhage by a dog in shock with the blood pressure at the critical level and that of a dog with the blood pressure at the same level from repeated hemorrhages. A dog weighing 13.6 Kg. was anesthetized with 0.035 Gm. of amytal per kilogram of body weight, and an arterial cannula was connected to a mercuric manometer for a continuous record of the blood pressure. Muscle pulp, 4.5 Gm. per kilogram of body weight, was then introduced into the peritoneal cavity. Nine hours later, when the mean blood pressure had declined to 72 mm. of mercury, the animal died within two hours from three bleedings of 10, 15 and 10 cc. respectively at intervals of thirty minutes.

In contrast, a dog weighing 11.6 Kg. was subjected to five repeated bleedings totaling 370 cc., or 3.2 per cent of the body weight, which reduced the pressure to 70 mm. Subsequently, five withdrawals of 20 cc. each, one of 15 cc. and eight of 10 cc. each at intervals of twenty to thirty minutes caused death. In other words, this dog withstood the loss of 195 cc. of blood during five hours after the blood pressure had been reduced to a critical level by previous hemorrhages. Death occurred thirty minutes after the last withdrawal of 10 cc. Hemodilution was progressive; at no time did the erythro-

TABLE 3.—*Comparison of Two Dogs*

Dog	Weight, Kg.	Blood Drawn, Percentage	Time, Hours	Hemodilution, Percentage
346 (not given anesthetic).....	10.0	9.1	31.5	77
373 (given amytal).....	10.8	4.48	31.0	32

cyte count of the hemoglobin content rise above the value noted in a previous examination. The results of the experiment are shown in figures 2 and 3.

The foregoing results illustrate an observation frequently made: that a person or an animal in shock may not survive a slight loss of blood which under other conditions would not be serious, and that hemorrhage when present is a contributory factor of highest importance.

LYMPH FLOW, TISSUE FLUID AND FLUID BALANCE

Some fifty years ago Heidenhain [3] observed that injections of diverse substances increased the flow of lymph. Other significant features of his report have escaped attention. He noted that the injections caused also a decline in the blood pressure and an increase in

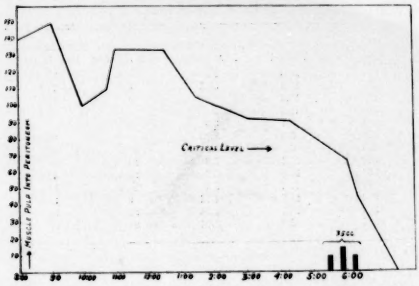


Fig. 2.—Blood pressure during shock. The scale at the left shows arterial pressure in millimeters of mercury. Loss of blood is indicated volumetrically by the height of the shaded columns; clock time is shown at the bottom. After the pressure had declined to 72 mm. three successive bleedings totaling 35 cc. caused death.

the total solids of the blood—hemoconcentration; the period of lowest blood pressure coincided with that of the maximal flow of lymph. Among the substances which produced these results were watery extracts of normal tissues, such as muscle, liver, spleen, kidney and mucosa, and bile, peptone and other secretions.

Subsequent studies (by Starling [4] and by Drinker and Field [5]) have shown that the increased flow of lymph, its high protein content, the decreased blood pressure and plasma volume and the hemoconcentration result from the leakage of plasma through capillary walls rendered abnormally permeable by the effects of the agents used. It has been shown that Heidenhain's "lymphagogues" and other substances which cause endothelial damage will produce the complete syndrome of shock if given in sufficient doses.

The following experiment illustrates the effect of shock on lymph flow.

A dog weighing 14 Kg. was placed under continuous ether anesthesia, the ether being administered through a tracheal tube, and the thoracic duct was cannulated. The rate of lymph flow was found to average 0.4 cc. per minute during a thirty minute period. The specific gravity of this lymph was 1.016, and it contained 420 cells per cubic millimeter. Seventy Gm. of muscle was excised from the dog's gluteal region, care being taken to avoid loss of blood by hemorrhage. The muscle was finely ground in a meat chopper, suspended in 150 cc. of physiologic solution of sodium chloride and introduced into the same dog's peritoneal cavity. During the ensuing two hours the rate of lymph flow averaged slightly more than 1 cc. per minute—an increase of 150 per cent. The specific gravity had risen to 1.018 and the cell content to 4,080 per cubic millimeter. The lymph became salmon colored, then pink, then finally bright red because of a progressive increase in the content of erythrocytes. Likewise, the coagulability of the lymph increased noticeably, so that it clotted frequently within the cannula, notwithstanding the increased rate of its flow. During four hours the hemoconcentration increased more than 20 per cent, as was shown by determination of the hemoglobin content and by counts of red cells. Similar changes in the flow and character of the lymph resulted when the experiment was repeated.

The effect of hemorrhage in another experiment performed under comparable conditions showed a striking contrast.

A dog weighing 21.8 Kg. was similarly anesthetized, and cannulation of the thoracic duct gave an average lymph flow of 0.66 cc. per minute during one hour. During the ensuing hour the dog was bled at intervals in amounts totaling 345 cc., 1.6 per cent of the body weight, or about 22 per cent of the estimated blood volume. During the next two hours the rate of lymph flow was only 0.21 cc. per minute—a decrease of 68 per cent. The lymph became lighter in color and did not coagulate spontaneously.

This substantial reduction in the flow of lymph and change in its composition are diametrically opposite to the change which accompanies shock. These results coincide with the observation of Starling that the formation and flow of lymph are decreased after hemorrhages.

The water content of various tissues was determined by weighing accurately the fresh, finely minced substance, desiccating it completely and weighing the residue to determine the water loss. Care was exercised in taking the specimen from the same area, e. g. the muscle or bowel, in each instance. Lung was not included in this comparison because the fluid content of the lobes varies and it was found technically difficult to desiccate the entire organ.

It was found that the skeletal muscles, liver and bowel wall contained more water after shock than after death by hemorrhages. Other investigators have recorded that these tissues become dehydrated after hemorrhages. Differences in the water content of the kidneys and pancreas were insignificant. An increase in tissue fluid was emphasized by Eppinger [6] as a regular occurrence in shock from trauma and from other causes. The fluid had a high protein content, and its chemical composition closely approximated that of plasma.

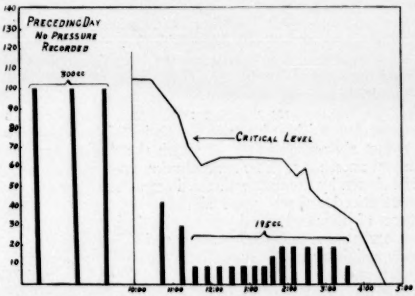


Fig. 3.—Blood pressure after hemorrhage. Three bleedings, producing 100 cc. of blood each, were done on the preceding day. After amytal anesthesia had been done the withdrawal of 70 cc. of blood brought the arterial pressure to 70 mm. A further loss of 195 cc. of blood, drawn in fourteen successive bleedings, was required to cause death.

Blalock reported the production of edema by hemorrhages, but we have not found confirmation of this in the observations of others. Physiologists agree in stating that hemorrhage is followed immediately by a

3. Heidenhain, R.: Versuche und Fragen zur Lehre von der Lymphbildung, Arch. f. d. ges. Physiol. **49**: 209, 1891.

4. Starling, E. H.: Principles of Human Physiology, Philadelphia, Lea & Febiger, 1936, p. 837.

5. Drinker, C. K., and Field, M. E.: Lymphatics, Lymph and Tissue Fluid, Baltimore, Williams & Wilkins Company, 1933.

6. Eppinger, Hans; Kaunitz, Hans, and Popper, Hans: Die seröse Entzündung, Berlin, Julius Springer, 1935.

transfer of fluid from the tissues to the blood. By this physiologic means for counteracting the effects of hemorrhage the lost blood volume is restored, but the water content of the tissues is reduced correspondingly. At necropsy the tissues are described regularly as ischemic and dry when uncomplicated hemorrhages have caused death.

A serious derangement of fluid balance is evidenced by the increased flow of lymph, the increase in tissue fluid, the hemoconcentration and the disturbance of absorption which accompanies shock. This derangement apparently has escaped the attention of most authors, for we have seen no discussion of it in the voluminous literature pertaining to the dynamics of shock. Several physiologic forces participate in maintaining normal relations between intravascular and extravascular fluids. But the action of these forces depends primarily and essentially on the presence of a semipermeable membrane between those fluids. Abnormal permeability of the endothelium throws out of gear the entire mechanism of fluid balance. The disturbance develops simultaneously with circulatory deficiency resulting from capillary injury, and it interrupts the process of absorption. Failure to absorb fluid from the tissues and from the gastrointestinal tract results in hemoconcentration—a characteristic sign of shock. Furthermore, the loss of fluid by vomiting, perspiration and diarrhea is not compensated readily. Fluids given intravenously leak out into the tissues, accentuating the edema without relieving the hemoconcentration.

Patients or animals affected by simple hemorrhages will absorb fluids from the tissues and from the gastrointestinal tract, thus restoring the blood volume. Suitable fluids given intravenously will be retained, because endothelial permeability has not been increased and the mechanism of fluid balance is not notably impaired.

COAGULABILITY

All the recorded observations we have found indicate that the coagulation time is appreciably lengthened during shock. This has been noted after burns, peptone poisoning, anaphylaxis and injections of histamine, as well as in the case of traumatic and experimental shock. Popielski [7] emphasized this feature and cited an instance in which blood from an animal in shock remained unclotted for sixty-four hours. Howell [8] commented on the low coagulability of the blood in anaphylaxis and after injections of protein cleavage products, peptone and histamine. He explained this as due to excessive mobilization of heparin from the liver. Perhaps the explanation applies also to delayed clotting in shock from other causes. We found the speed of coagulation noticeably retarded in 10 of 12 dogs tested during shock. One animal showed more rapid coagulation, and 1 showed no significant change.

After hemorrhages the formation of fibrin is accelerated and the clotting time is shortened.[9] The acceleration is detectable within ten minutes after the hemorrhage.[10] In our experiments, the blood of dogs clotted normally in eight to twelve minutes. After each successive bleeding the coagulation was more rapid, and when the blood pressure had declined to the critical level the blood clotted completely in one to four minutes. A decided increase in the speed of coagulation occurred after hemorrhages in 9 of 10 dogs on which observations were made. In 1 dog no significant change occurred.

BLOOD CHEMISTRY

An increase in the nonprotein nitrogen content of the blood during shock was first reported by Duval and Grigaut [11] (1918). This was confirmed by Aub and Wu and by others. Whipple and Van Slyke [12] found that the increase consisted largely of urea, amino and peptid nitrogen. Other investigators have shown that the creatinine content is also increased. These changes occur with traumatic shock, burns, intestinal obstruction and anaphylaxis and after injections of commercial peptone and of histamine. Lurje [13] found an increased nonprotein nitrogen content in the blood during shock after surgical operations in 8 patients. The level returned to normal when the circulatory deficiency was relieved. Whipple attributed the rapid piling up of nitrogen in the blood to a rapid breakdown of protein substances and to retarded renal elimination. It is recalled that severe disturbance of renal function, indicated by oliguria and by albumin, bile, débris, casts and erythrocytes in the urine, frequently accompanies shock (Eppinger [6]).

An increase in the hydrogen ion concentration and a decrease in the carbon dioxide–combining power of the plasma have long been recognized as regular features. These were considered etiologic until the results of experimentation showed them to be effects rather than causes.

Hypochloremia, chiefly a diminution of the level of sodium chloride, has been shown to accompany the increased nonprotein nitrogen content and decreased alkaline reserve mentioned previously. Scudder [14] has shown that the blood sodium content is decreased in shock from various causes. His summary on changes in the blood indicated also that the potassium, calcium and magnesium contents are increased and that the amount of phosphates is decreased.

Isolated reports have indicated that the alkaline reserve is decreased and the chloride levels increased after hemorrhages. Scudder's data indicated that the blood potassium level is elevated during the moribund state resulting from loss of blood. Leichsenring and Biester's [15] studies on the effects of hemorrhages in dogs showed a decrease in the nonprotein nitrogen level on the first day after the hemorrhages in eight of ten tests and a slight increase in two tests. They said: "The nonprotein nitrogen level per unit volume was slightly below normal during the postbleeding period." They cited reports of somewhat variable results by other workers and regarded the changes in the nonprotein nitrogen level after the loss of blood as transitory.

We undertook a comparison of the chemical and other hematologic changes which occur during shock and after hemorrhages. In the earlier tests, data on the

7. Popielski, L.: Ueber die physiologische Wirkung von Extrakten aus samtlichen Teilen des Verdauungskanales, Arch. f. d. ges. Physiol. **128**: 191, 1909.

8. Howell, W. H.: Address: Recent Advances in the Problem of Blood Coagulation Applicable to Medicine, J. A. M. A. **117**: 1059 (Sept. 27) 1941.

9. Best, C. H., and Taylor, N. B.: Physiological Basis of Medical Practice, Baltimore, Williams and Wilkins Company, 1939, p. 36.

10. Castle, W. B., and Minot, G. R.: Pathologic Physiology and Clinical Description of the Anemias, New York, Oxford University Press, 1936, p. 36.

11. Duval, P., and Grigaut, A.: La rétention azotée des blessés, Compt. rend Soc. de biol. **81**: 873, 1918.

12. Whipple, G. H., and Van Slyke, D. D.: Proteose Intoxication and Body Protein, J. Exper. Med. **28**: 213 (Aug.) 1918.

13. Lurje, A.: Protein Metabolic Disturbances Under Conditions of Shock and Surgical Interference, Am. J. Surg. **32**: 313 (May) 1936.

14. Scudder, J.: Shock: Blood Studies as a Guide to Therapy, Philadelphia, J. B. Lippincott Company, 1940.

15. Leichsenring, Jane M., and Biester, Alice: The Blood Picture in Hemorrhagic Anemia, Technical Bulletin 139, University of Minnesota, Agricultural Experiment Station, 1939.

blood of dogs at or immediately before death were compared with data on the same dogs before the experiments. Then it was realized that values derived from moribund animals are not so reliable and may show variations not present before the animals were in a dying condition. In order to obviate this source of error in subsequent experiments, blood was drawn for examination when hemoconcentration or hemodilution indicated that the circulation was seriously affected by shock or by hemorrhage respectively. When a continuous record of the blood pressure was made, blood for chemical tests was drawn when the pressure had declined to a critical level of 70 mm. of mercury. In 4 dogs shock was induced by absorption of muscle pulp from the peritoneal cavity, in 2 by repeated subcutaneous injections of histamine and in 1 by anaphylaxis (from horse serum). The data from these comparisons are shown in tables 5 and 6.[16]

A comparison of these data indicates that hemoconcentration, as shown by erythrocyte counts and hemoglobin and specific gravity readings, occurred regularly during shock and that hemodilution resulted after bleedings.

There was a noticeable increase in the nonprotein nitrogen content during shock in each instance; a moderate decrease was found in 4 of 6 dogs after hemorrhages. In 2 there was a slight increase.

The chloride content of the plasma declined moderately in 5 of 7 dogs during shock; in 2 there was no significant change. The level of chlorides increased in 5 of 6 dogs as a result of bleedings.

TABLE 5.—*Changes in the Blood During Shock*

Cause of shock		Dog 371 Muscle pulp	Dog 374 Muscle pulp	Dog 376 Muscle pulp	Dog 378 Hista-mine	Dog 379 Anaphy-laxis	Dog 380 Hista-mine	Dog 384 Muscle pulp
Erythrocytes, millions	Before	4.86	5.76	5.78	5.94	6.33	6.92	6.0
	After	6.01	6.45	6.32	7.26	8.17	8.82	7.62
Hemoglobin, Gm.	Before	10.37	14.74	13.8	11.1	13.44	14.64	11.1
	After	11.9	16.00	16.2	12.84	16.56	16.56	15.1
Specific gravity	Before	1.049	1.034	1.060	1.030	1.037	1.030	1.032
	After	1.065	1.070	1.068	1.054	1.061	1.058	1.058
Nonprotein nitrogen	Before	21.6	32.85	32.13	23.0	14.2	21.84	17.13
	After	43.2	40.0	65.26	56.0	18.46	67.86	34.72
Chlorides	Before	369	380	390	362	360	377	370
	After	371	379	379	337	355	343	371
Serum protein	Before	8.44	5.4	5.42	5.85	8.53	5.0	5.57
	After	7.04	4.7	4.62	4.92	4.20	3.99	3.69
Blood sugar	Before	82.5	146.0	128.75	116.0	127.68	119.52	113.16
	After	283.8	127.0	108.68	225.0	142.5	209.28	71.34
Carbon dioxide, volume per cent	Before	43.67	46.11	56.86	50.6	47.5	55.6	53.87
	After	41.77	39.52	13.76	30.78	40.78	37.2	47.26

TABLE 6.—*Changes in the Blood After Hemorrhage*

		Dog 372*	Dog 373*	Dog 375*	Dog 377	Dog 382	Dog 383
Percentage of body weight bled		4.78	4.48	3.14	5.6	5.1	6.8
Time		6 hrs.	31 hrs.	25 hrs.	8 hrs.	22 hrs.	26 hrs.
Erythrocytes, millions	Before	5.500	5.380	5.410	5.270	5.670	6.190
	After	2.360	2.790	1.990	1.090	2.050	2.170
Hemoglobin, Gm.	Before	13.0	14.03	14.07	13.56	10.40	11.6
	After	8.54	9.57	6.40	6.75	4.80	4.8
Specific gravity	Before	1.050	1.025	1.051	1.044	1.038	1.044
	After	1.037	1.018	1.026	1.027	1.018	1.024
Nonprotein nitrogen	Before	28.6	30.58	25.00	18.56	25.5	31.5
	After	21.1	31.75	20.70	30.7	18.9	22.63
Chlorides	Before	358	374	391	386	360	374
	After	326	414	416	405	378	380
Serum protein	Before	5.53	4.38	4.60	5.06	5.06	4.68
	After	4.3	3.44	3.65	4.10	3.91	3.89
Blood sugar	Before	140	141	143.5	120.00	123.1	104.4
	After	189	184	167.5	646.75	243.6	392.3
Carbon dioxide, volume per cent	Before	50.83	43.80	62.01	51.36	52.4	55.0
	After	51.77	34.48	43.53	36.68	50.4	30.25

* Dogs 372, 373 and 375 were under amytal anesthesia during part of the experiment.

There was an appreciable decline in the content of serum proteins per hundred cubic centimeters and in the carbon dioxide content of the plasma both in shock and after hemorrhage. The blood sugar content varied irregularly during shock, but a considerable increase occurred regularly after hemorrhage. Hence total proteins, blood sugar and reserve alkalinity are not points of distinction between shock and hemorrhage.

COMMENT

Several workers [17] reduced the blood pressure of dogs below 70 mm. of mercury by the repeated withdrawal of blood from the femoral artery. When the arterial pressure sank, blood was introduced by direct transfusion;

16. Chemical determinations of the blood or blood serum were made according to the following methods: the Koch and McMeekin for nonprotein nitrogen, the Folin and Wu for sugar, the Whitehorn for chlorides, the Peters and Van Slyke for carbon dioxide content and the Looney and Walsh for total protein. Descriptions of these methods may be found in:

Koch, F. C., and McMeekin, T. L.: A New Direct Nesslerization Micro-Kjeldahl Method and a Modification of the Nessler-Folin Reagent for Ammonia, J. Am. Chem. Soc. **49:** 2066 (Sept. 5) 1924.

Folin, O., and Wu, H.: A System of Blood Analysis: I. A Simplified and Improved Method for Determination of Sugar, J. Biol. Chem. **41:** 367, 1920.

Whitehorn, J. C.: Simplified Method for Determination of Chlorides in Blood or Plasma (Supplement to Folin's System of Blood Analysis), J. Biol. Chem. **45:** 449, 1921.

Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1932, vol. 2, pp. 245-251.

Looney, J. M., and Walsh, A. L.: The Determination of Globulin and Albumin in Blood Serum by the Photoelectric Colorimeter, J. Biol. Chem. **130:** 635 (Oct.) 1939.

17. Blalock, Alfred: Principles of Surgical Care, Shock and Other Problems, St. Louis, C. V. Mosby Company, 1940. Freeman, N. E.; Shaffer, S. A.; Schecter, A. E., and Holling, H. E.: The Effect of Total Sympathectomy on the Occurrence of Shock from Hemorrhage, J. Clin. Investigation **17:** 359 (May) 1938.

when it rose above 70 mm., a quantity of blood was again removed. By this combination of removal and reintroduction of blood, the pressure was maintained at about 70 mm. for several hours. The animals showed a number of the clinical features of shock: weakness, pallor, thready pulse and low blood pressure. Hemo-

TABLE 7.—*Items Identical in the Two Conditions*

Sympathoadrenal activity
Stimulation of myocardium
*Strong, rapid pulse in early stages
*Peripheral vasoconstriction
Reduced volume flow
*Peripheral ischemia, pallor
*Loss of tissue turgor
Discharge of reservoir blood into systemic circulation
*Contraction of spleen
*Increased blood sugar content
*Dilatation of pupils, often perspiration
Low basal metabolism
*Declining temperature
*Decreased alkaline reserve
*Decreased serum protein content
*Increased respiratory rate, thirst
*Low arterial blood pressure (in late stages)
*Death due to inadequate circulatory function

concentration was observed in all such experiments. "Particularly impressive is the finding that all animals died despite the fact that more blood was introduced than had been removed." In other words, transfusion was without benefit after low blood pressure had existed for an extented period. Examination of the tissues after death showed visceral congestion, edema and petechial hemorrhages. "These observations prove that shock can be produced by uncomplicated hemorrhage and that hemoconcentration, a negative response to transfusion and alterations in the tissues result if the circulation remains depressed for several hours."

Before accepting these deductions in toto, one should examine thoughtfully the conditions of the experiments. It is known that neither man nor animals can survive when the arterial pressure is maintained, by whatever means, below the "critical level," i. e. 70 to 80 mm. of mercury.

Certainly when the blood pressure is maintained at 70 mm. continuously by hemorrhage or by any other means a point will be reached after which neither transfusion nor any other procedure will restore circulatory efficiency. Animals so treated inevitably die. When an animal is dying from any cause there is relaxation of vascular tonus affecting particularly the capillaries and venules. Anoxia, which regularly accompanies the moribund state, causes abnormal permeability of the endothelium. If at this stage a quantity of blood is introduced it will distend the atonic vessels and will produce visceral congestion. If death does not occur immediately, leakage of plasma will cause both edema and hemoconcentration.

If transfusions of blood were given to a dog moribund from some natural cause, such as neoplasm, infection with worms or nephritis, the same results would be expected to follow. Would the negative response to transfusions, unremitting decline in blood pressure, visceral congestion, edema and hemoconcentration indicate that neoplasm or other disease caused shock?

Other readers may interpret such results as the artificial production of the features mentioned and as a demonstration that vascular atony and endothelial permeability occur whenever an animal is moribund.

We have been unable to demonstrate hemoconcentration and the visceral pathologic changes of shock in animals brought to death by repeated losses of blood. Nevertheless, we believe that capillary atony and endothelial permeability develop as terminal events when an animal is moribund from hemorrhages or from any other cause. These changes are accompanied by a grave derangement of fluid balance.

A highly important difference between shock and the effects of hemorrhages, as we see it, is that endothelial injury and the resulting disturbances of circulation and of fluid balance occur as initial derangements causing shock, while the same disturbances develop only in the terminal or moribund state after hemorrhages.

SUMMARY

Several features in the syndrome of shock occur also after hemorrhage because the two evoke the same physiologic mechanism for counteracting the disturbance. The points of resemblance are arranged in table 7. The items which we have verified experimentally are marked with an asterisk.

Incipient circulatory deficiency from shock, hemorrhage or other causes evokes compensatory reactions by the sympathoadrenal system resulting in identical responses. Hence no distinctions from the effects of hemorrhage were evident, and the two conditions were regarded as identical.

We have assembled the various physiologic and biochemical features which have been reported in cases of shock from wounds, burns, intestinal obstruction, anaphylaxis and other causes and reports on the same items as observed after uncomplicated hemorrhage.

TABLE 8.—*Comparison of Results of Hemorrhage and Shock*

Items	Shock	Hemorrhage
*Endothelium	Permeable to colloids	Impermeable to colloids
*Flow of lymph	Increased	Decreased
*Tissue fluid	Increased	Decreased
*Fluid balance	Disturbed	Undisturbed
*Pulmonary edema	Frequent	Absent
*Absorption from gastrointestinal tract	Impaired	Unimpaired
*Absorption from tissues	Impaired	Unimpaired
*Vomiting	Persistent	No vomiting
*Diarrhea	Frequent	Absent
*Effect of moderate loss of blood	Severe	Mild
Transfusion of blood	Often ineffective	Effective
*Saline solution, administered intravenously	Ineffective	Often effective
*Urine	Concentrated, low volume; contains albumin, erythrocytes, bile and débris	No characteristic changes
Blood		
*Coagulation time	Lengthened	Shortened
*Hemoglobin content, erythrocyte count	Increased	Decreased
*Specific gravity	Increased	Decreased
*Nonprotein nitrogen level	Increased	Decreased
Potassium content	Increased	Terminal increase
Calcium content	Increased	Decreased
Magnesium content	Increased	Decreased
*Chloride content	Decreased	Increased
Changes Seen at Necropsy		
*Edema of soft tissues	Characteristic	None
*Serous effusions	Present	Absent
*Capillovenous congestion	Characteristic	Absent
*Petechiae	Characteristic	Absent
*Visceral ischemia	Absent	Present
Weight of organs	Increased	Decreased
*Gastrointestinal tract	Dilated, atonic	Contracted
*Parenchymal necroses	Present	Absent

These have been arranged in table 8 to facilitate comparison. We have verified experimentally the items marked with an asterisk.

Medical history records instances in which a symptom complex, once regarded as a disease entity, later has been shown to comprise several more or less unrelated conditions. The terms leprosy and Hodgkin's disease originally were applied to groups of conditions

having clinical resemblances but later shown not to be identical. Few terms have been used more loosely and indefinitely than shock. Hence it is not strange that the effects of hemorrhage, which clinically resemble shock so closely, should have been included without discrimination in the broad application of that term.

It is remarkable that numerous important differences so long have escaped the attention of persons interested in these conditions. We humbly admit our own error in subscribing for a time to the belief that uncomplicated hemorrhage will produce the syndrome of shock.

Since this article was written, Price [18] and his associates have published the effects of simple hemorrhages as observed in 37 dogs. Their results confirm those which we have recorded. The interval between the first bleeding and the death of the animals ranged between five and six hours. Hemodilution occurred regularly; the clotting time was reduced; the plasma chlorides and sodium increased; the potassium and the nonprotein nitrogen of the blood rose shortly before death. Necropsy showed pallor of the tissues and organs. No edema nor increased wetness of the tissues, no abnormal fluid in serous cavities, no visceral congestion nor evidence of increased capillary permeability was seen. They concluded that "Evidence is presented to show that capillary atony, 'stasis' and abnormal permeability of capillaries probably do not occur in dogs following uncomplicated acute hemorrhage."

CONCLUSIONS

Points of similarity between shock and the effects of hemorrhage result in part from the fact that each evokes the same mechanism of compensation and in part from the deficiency of circulation which each produces.

Points of contrast between shock and the effects of hemorrhage are so numerous that the assumed identity of those conditions is no longer tenable.

Hemorrhage when present is a highly important contributory factor because the derangement of fluid balance which accompanies shock interrupts the processes by which loss of blood is compensated.

For the sake of accuracy the expression shock and hemorrhage should be substituted for the term hemorrhagic shock.

We urge investigators not to employ hemorrhage as a means for producing shock experimentally. If the results so obtained are interpreted as applying to shock, erroneous conclusions may be drawn.

1025 Walnut Street.

ABSTRACT OF DISCUSSION

DR. HENRY H. HARKINS, Detroit: Dr. Moon and I represent almost opposite extremes of belief concerning the mechanism of shock, especially concerning the particular aspect that Dr. Moon and his co-workers have presented today. One of the differences between our views is: Are hemorrhage and shock the same? At no time have I stated that hemorrhage is the same as shock, but I do believe that hemorrhage will lead to shock. Dr. Moon states that transfusions are effective in hemorrhage but not in shock. My work and that of Dr. Blalock and others have led to a contrary conclusion, namely that, if the hemorrhage is allowed to continue long enough, the restitution of even more blood than was removed will not restore the animal or patient. I am sure all of you have had experience with instances of severe bleeding that come in late and where large blood transfusions are given (probably larger than the amount of blood that was lost) and yet the patient does not recover. In the authors' chart where they discussed 26 dogs with protracted hemorrhage so that the dogs did not die for a long period, I should like to know whether or not he had blood pressure readings. Several authors have found that pathologically the changes in protracted hemorrhage are much the same as in protracted shock. However, it is essential that the blood pressure remain low for several hours if hemorrhage is to cause these pathologic changes. In such cases the pathologic changes at autopsy will be much the same whether the shock was produced by hemorrhage or by other injury, as shown by Blalock of Johns Hopkins, the late Mr. O'Shaughnessy of London, and more recently by Dunphy, Gibson and Keeley. From the practical point of view, especially when we are approaching war, it should be pointed out that, even if shock produced by other means differs somewhat from shock produced by hemorrhage, too much importance should not be attributed to this from the point of view of treatment. Since we do not know about some of the obscure manifestations of shock and our chief means of treatment is to give fluid replacement, whether the fluid lost is whole blood or plasma, the most important item is to restore it as quickly as possible. If a patient has hemorrhage plus other types of shock there may not be hemoconcentration. Some of my recent work not yet completed has shown that in shock a great deal of fluid is lost remote from the site of injury, in agreement with the work of Moon and his collaborators.

18. Price, P. B.; Hanlon, C. R.; Longmire, W. P., and Metcalf, William: Effects of Acute Hemorrhage in Healthy Dogs, Bull. Johns Hopkins Hosp. **69**: 327 (Oct.) 1941.

THE SURGICAL TREATMENT OF PROSTATIC DISEASE

ROY B. HENLINE, M.D.
NEW YORK

Surgical diseases of the prostate gland present so many variations that no one surgical procedure will obtain the best results in all cases. A thorough understanding of the site of origin and progression of the disease permits one to select the proper surgical approach in each instance. So commonly do surgeons refer to the various types of prostatic operations as "removal of the prostate" that many forget that the prostate is rarely actually removed. In many instances only the hyperplastic tissue, or so-called adenoma, is removed; the functioning prostatic glands or their remnants remain firmly attached to the surrounding fascia and together are referred to as the false capsule of the prostate. Often removal of this hyperplastic tissue is all that is required to establish adequate drainage of the bladder, and the remaining compressed, uninvolved prostatic tissue may reasonably be left behind. It is not uncommon, however, for these remaining prostatic glands or their remnants to be the seat of a pathologic process which also requires removal for the patient to be cured.

ETIOLOGY

There is evidence that hyperplasia (adenoma) of the prostate in advancing years is incited by an imbalance of male and female hormones. As yet, however, there is no pathologic evidence that hormonal therapy will inhibit or stop this process once it has begun.

Just which elements in the prostate give rise to prostatic hyperplasia has been a much discussed subject for years, although observers have agreed that the new growth makes its first appearance near the urethra and just under the mucosa. Tandler and Zuckerkandl,[1]

Read before the Section on Urology at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 5, 1941.

The expenses of this research were defrayed from the Alfred Busiel Research Fund of the Department of Urology, James Buchanan Brady Foundation, of the New York Hospital.

1. Tandler and Zuckerkandl: Studien zur Anatomie und Klinik der Prostatahypertrophie, Berlin, 1922, cited by Young, H. H., and Davis, D. M.: Practice of Urology, Philadelphia, W. B. Saunders Company, 1926, vol. 1, chapter 7.

among others, expressed the belief that prostatic hyperplasia develops from the short prostatic glands which extend only a short distance beneath the mucosa in the prostatic portion of the urethra (fig. 1). This is the locality where hyperplastic changes are first seen in the prostate. Deming and Wolf,[2] in a recent study of 210 prostates, agreed with these authors regarding the location of the new growth but claimed that hyperplasia does not arise directly from these short prostatic glands, being first seen as a fibromuscular mass in the muscular wall of the prostatic portion of the urethra. Deming [3] suggested that this mass may correspond to uterine myoma, since theoretically the two growths have a common embryologic anlage. The fibromuscular masses lie parallel to the muscular fibers of the urethra between the prostatic ducts and stimulate epithelial hyperplasia from these ducts of the prostatic glands (fig. 2). Deming and Wolf stated that this hyperplasia is not an enlargement of existing prostatic glands but, although similar in structure, is composed of newly formed glandular elements from the ducts of the prostatic glands. This explains its locality surrounding the prostatic urethra, from which the growth extends to compress the true lateral and posterior lobes toward the capsule and hence assumes the position of the lateral lobes on each side of the urethra (figs. 3 and 4).

Hyperplasia of the prostate may occur just under the urethral mucosa in the region of the lateral lobes, the median commissural lobe, the subcervical lobes or the subtrigonal lobes (fig. 5). However, it does not occur in the posterior lobe of the prostate, nor does it originate in the functioning glands of the lateral or median prostatic lobes. As the hyperplasia in the region of the lateral lobes increases in size, the prostatic portion of the urethra is distorted with an anteroposterior elongation (figs. 3 and 4). Eventually the urethra is so compressed as to cause urinary retention.

Since the ducts of the prostatic glands are involved in this hyperplasia, the drainage from these glands into the prostatic portion of the urethra is gradually obstructed. If the glands are infected, a walled-off focus of infection may remain in the compressed remnants of the prostate gland.

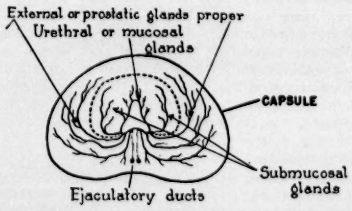


Fig. 1.—Prostatic hyperplasia develops near the urethra, as shown diagrammatically by the encircled dotted line. The external glands, or prostatic glands proper, are compressed toward the capsule as the hyperplastic mass increases in size. The posterior lobe is not shown but is in the region marked "ejaculatory ducts."

PROSTATIC CALCULI

The entire prostate undergoes a change at the age of about 50. R. A. Moore [4] has shown how poor drainage from the prostatic glands predisposes to the formation of a nucleus of desquamated epithelium and débris to form corpora amylacea. These foreign bodies are formed in the functioning prostatic glands in contradistinction to the hyperplasia, which develops near the urethra from the gland ducts. The corpora amylacea are frequently surrounded by round cell infiltration and

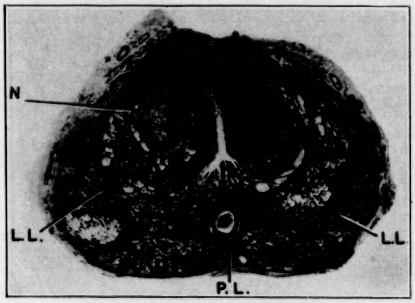


Fig. 2.—Cross section (× 2) of prostate of a man aged 74 showing nodule (*N*) one half of which is glandular and the other half aglandular. Note fibromuscular masses on each side of the urethra being invaded by epithelial hyperplasia from the ducts of the prostatic gland. Note beginning compression of the lateral lobes (L. L.) by this mass and the uninvolved posterior lobe (P. L.). (From Deming.)

fibrosis in the prostatic gland itself. Calcium salts may be deposited around these nuclei to form prostatic calculi.

Corpora amylacea, or prostatic calculi, often surrounded by evidence of infection, may be present for years without causing any symptoms. In some patients, however, a severe infection develops and symptoms are produced that require surgical intervention. In other patients hyperplasia (adenoma) may develop in the region of the ducts near the urethra. As this increases in size the corpora amylacea, infection and calculi if present are compressed toward the capsule with the prostatic glands; the ducts from the prostatic glands are gradually occluded, and the infected prostatic remnants may no longer drain into the urethra. Some glands become atrophic, while in others infection or calculi may be walled off by the enlarging hyperplasia (fig. 6).

CARCINOMA

Carcinoma of the prostate is said to originate in the posterior lobe in about 75 per cent of cases. Recent statistics by Moore [5] and Rich [6] drew attention to the fact that between 14 and 16 per cent of all men over 50 have prostatic carcinoma. Although these percentages seem rather high, carcinoma of the prostate either is becoming more common or is being recognized more frequently. In treating prostatic disease one cannot overlook these observations without giving serious thought to rational treatment. Since the average life expectancy of men has increased, it may be that one should expect to find prostatic malignant neoplasms more frequently.

DIAGNOSIS

The exact nature and extent of prostatic disease should be determined before the type of surgical procedure to be employed is decided on. Frequent urina-

2. Deming, C. L., and Wolf, J. S.: The Anatomical Origin of Benign Prostatic Enlargement, J. Urol. **42**: 566-580 (Oct.) 1939.
3. Deming, C. L.: The Development of Prostatic Hyperplasias, Surg., Gynec. & Obst. **70**: 588-594 (Feb.) 1940.
4. Moore, R. A.: Morphology of Prostatic Corpora Amylacea and Calculi, Arch. Path. **22**: 24-40 (July) 1936.
5. Moore, R. A.: Evolution and Involution of Prostate Gland, Am. J. Path. **12**: 599-624 (Sept.) 1936.
6. Rich, A. R.: On the Frequency of Occurrence of Occult Carcinoma of the Prostate, J. Urol. **33**: 215 (March) 1935.

tion and the finding of residual urine in the bladder and of an enlarged prostate by rectal examination are not sufficient evidence to suggest the employment of a particular type of surgical procedure. Rectal examination, though valuable, may be misleading in the diagnosis of the presence or the extent of prostatic disease in almost 50 per cent of cases.[7] It is not uncommon

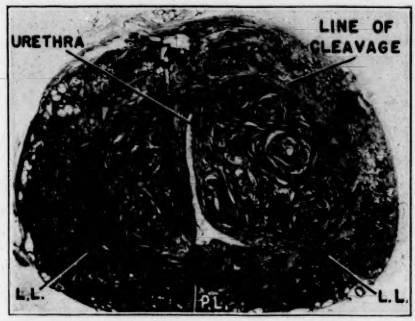


Fig. 3.—Cross section (under low power) of an entire prostate gland, showing multiple adenoma in neighborhood of urethra. Periphery shows nonadenomatous prostatic tissue. Note the replacement of the fibromuscular mass by hyperplasia, which is assuming the position of the lateral lobes. The lateral and posterior lobes are further compressed and are becoming functionless. Between the hyperplasia and the compressed lateral lobes is the "line of cleavage" where "enucleation" of the prostate by suprapubic or transurethral prostatectomy is performed, the remnants of prostate left behind being called the false capsule. (From Geraghty.)

to have a patient who has been referred by a physician state that rectal examination has ruled out prostatic disease, only to find that the prostate is actually the source of his symptoms. Hyperplasia of the subcervical or subtrigonal lobes or of the median commissural lobe and the presence of fibrous bars at the vesical outlet are among the conditions which may cause no change in the contour or size of the prostate as revealed by rectal palpation yet may require surgical intervention for their relief.

The exact location and extent of the obstruction should be established. The presence of prostatic infection, fibrosis or calculi should be determined. Cystoscopic examination when indicated will aid the urologist in selecting the type of operation in many instances. The presence of a hard nodule in the prostate determined by rectal palpation is suggestive of a malignant neoplasm, and the nodule should be treated as such until proved otherwise.[8] Considerable information concerning the size and extent of the prostatic obstruction may be obtained by an aerogram and cystourethrogram, with the patient in a semilateral position. In most instances the procedure may be performed with little discomfort to the patient.[9]

TREATMENT

When the exact diagnosis of the type of prostatic disease has been established, one must decide on the surgical procedure which will relieve the patient promptly and permanently. The origin and location of the disease and its underlying pathologic changes should be considered, since a clear understanding of the progression of the disease is often the deciding factor between one approach and another. To attempt to remove a fibrous prostate, particularly if calculi are present, by suprapubic prostatectomy reveals poor surgical judgment. One cannot remove the posterior lobe of a prostate containing an early malignant neoplasm by suprapubic prostatic enucleation.

The particular method used for removal of the prostate has, in the past, depended to a large degree on the choice of the surgeon, who has considered only his training and ability to relieve the obstruction. In some instances he has overlooked the underlying pathologic condition and has not selected the operation most likely to give the patient complete and permanent relief from his prostatic disease. In other words, the surgical procedure employed should depend, not on the surgeon's skill with any one method, but on the nature of the pathologic process in the prostate.[10]

It is essential that renal damage, infection and hemorrhage and retention of urine be considered in the treatment of all patients with prostatic disease. These conditions have been thoroughly discussed in the literature. One finds statistics showing a low mortality for each type of surgical approach to the prostate, and one is aware that the low mortality is the result of the proper preparation of patients by a surgeon who is highly skilled in the surgical approach he advocates. With the same patients and a surgeon skilled in a different surgical approach to the prostate, a similar mortality would probably result.

In some patients who have had a suprapubic enucleation of the prostate or a transurethral resection, a postoperative morbidity persists, with infected urine and frequent urination. These symptoms are distressing both to the surgeon and to the patient. Sometimes they result from infection in the remnants of the prostatic tissue or the seminal vesicles and might have been prevented by removal through the perineum of the prostatic capsule and prostatic remnants together with the hyperplasia.

Transurethral removal of obstructing hyperplastic tissue from the prostate is a rational procedure and has assumed an important place in prostatic surgery. With proper selection of cases and a skilled surgeon, excellent results can be obtained. Opinions vary as to the size of the prostate which is best treated by transurethral operation. However, with a surgeon properly skilled in this procedure, relatively large hyperplastic masses may be adequately removed. Some surgeons prefer to remove large intravesical prostatic hyperplastic masses suprapubically, and similar results may be obtained with these growths by either method.

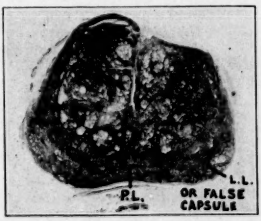


Fig. 4.—Cross section of prostate removed by subtotal perineal prostatectomy. Note progression of hyperplasia and further compression of functioning prostatic glands which now have become the false capsule. Enucleation would not remove these prostatic remnants. The line of cleavage between the hyperplasia and the compressed prostatic remnants is clearly visualized.

7. Henline, R. B.: Prostatic Disease: Determination of Method of Treatment, New York State J. Med. **38**: 773-787 (May 15) 1938. Young and Davis.[3]

8. Hinman, Frank: The Perennial Dispute in the Treatment of Prostatism, J. A. M. A. **112**: 424 (Feb. 4) 1939.

9. Henline, R. B.: Prostatic Disease with Special Reference to the Various Causes and Types as Well as Their Treatment, South. Surgeon **9**: 360-368 (May) 1940.

10. Henline, R. B.: The Adequate Treatment of Prostatic Disease with Special Reference to Pathology, New York State J. Med., to be published.

When it becomes desirable to remove the remnants of the functioning prostatic glands because of infection, calculi or a hard nodule in the prostate suggestive of a malignant neoplasm, it is essential to approach the prostate by the perineum.[11] Neither by the transurethral nor by the suprapubic approach is it possible to remove either these prostatic remnants or the posterior

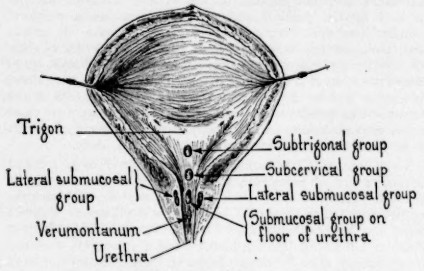


Fig. 5.—Localities where hyperplasia may develop.

lobe of the prostate. The posterior lobe and the false prostatic capsule are separated from the hyperplastic mass in the prostate by a line of cleavage which permits the hyperplastic tissue to be cleanly "shelled out" suprapubically (fig. 3). The remnants of the prostate, in which it is estimated that 75 per cent of carcinoma develops (posterior lobe), must of necessity be left behind in either a suprapubic enucleation or a transurethral resection.

SUBTOTAL PERINEAL PROSTATECTOMY

New perineal operations have recently been devised.[12] Among these is a subtotal perineal prostatectomy devised by me primarily for the complete removal of prostatic calculi.[11] Results with this procedure have been so satisfactory that its use has been extended to the treatment of severe prostatic infection with hyperplasia, prostatic fibrosis in some cases, all early malignant neoplasms and extensive hyperplasia of the lateral lobe in some cases.

The method consists in the usual perineal exposure and removal of the entire prostate, capsule and prostatic portions of the urethra. Hemorrhage is controlled by sutures. The distal portion of the urethra is sutured around a catheter to the neck of the bladder and the prostatic portion of the urethra is thus entirely eliminated.[11]

By this subtotal method[13] the obstructing tissue as well as all remnants of compressed prostatic tissue may be completely removed. No focus of infection remains in the prostatic portion of the urethra to cause persistent urinary symptoms. The posterior prostatic lobe is removed, and hence a possible source of undiagnosed malignant growth is eliminated. The patient should be told that temporary or even permanent sexual impotence may result from this procedure.

When a suspiciously hard nodule is palpated in the prostate on rectal examination a perineal exposure, with complete removal of the prostate, offers the only hope of cure should the growth prove to be early carcinoma of the prostate. The posterior lobe of the prostate can be reached only by the perineal approach, and the patient deserves to be operated on by the method which offers him a hope of cure. When it is considered that 1 of every 5 to 7 patients past the age of 50 has carcinoma of the prostate, is one not justified in removing the posterior lobe of the prostate, together with the obstructing portion of the gland, when the condition is suspected? Other methods of treating prostatic carcinoma are rarely curative and offer only relief from symptoms.

It is my custom to expose the prostate by opening the perineum when a suspicious nodule has been palpated. A small section of the suspected area is removed and a frozen section made at once. If carcinoma is discovered a radical perineal prostatectomy is performed, the entire prostatic portion of the urethra and the seminal vesicles being included. If biopsy proves the growth benign, either a conservative prostatectomy is performed, if indicated, or the perineum is closed. Exposure of the prostate by opening the perineum, with performance of biopsy, is far superior to repeated attempts to obtain a specimen with a needle through the perineum. A negative report on such a specimen proves nothing, since the involved area may be small and easily missed. In a recent case, taking a small biopsy by perineal exposure of the prostate removed the entire malignant area, and careful microscopic examination of the rest of the prostate and the seminal vesicles failed to reveal any sign of malignancy.

Since cancer of the prostate is so common, and until such time as conservative treatment of prostatic carcinoma proves more satisfactory as to cure and the prevention of the subsequent suffering, it may not be unreasonable to consider the advisability of removing the posterior prostatic lobe and capsule when surgical intervention must be instituted anyway for obstruction. Although I do not recommend this procedure routinely, the conclusions drawn from statistics may force the

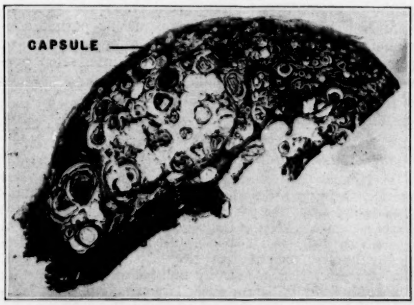


Fig. 6.—Prostatic calculi. Note the presence of calculi throughout the functioning prostatic glands, extending to the outside surface of the capsule.

consideration of it in the future if patients are to be offered the best outlook for continued health.

I am opposed to the pessimism frequently voiced regarding carcinoma of the prostate. For a physician to palpate a suspiciously hard nodule in the prostate and "wait" to see what happens is a violation of the trust placed in him. It is only patients with such nodules that one can hope to cure of this disease, and

11. Henline, R. B.: Prostatic Calculi: Treatment by Subtotal Perineal Prostatectomy, J. Urol. **44**: 146 (Aug.) 1940.
12. Belt, Elmer; Ebert, C. E., and Surber, A. C., Jr.: A New Anatomic Approach in Perineal Prostatectomy, J. Urol. **41**: 482-497 (April) 1939. Gibson, T. E.: Improvements in Perineal Prostatectomy, Surg., Gynec. & Obst. **47**: 531.539 (Oct.) 1928.
13. "Subtotal method" refers to removal of the entire prostate with the capsule and all prostatic tissue and is distinguished from radical total prostatectomy since the seminal vesicles are allowed to remain intact.

unfortunately most of them have no urinary symptoms! In the greater number of cases of prostatic carcinoma, when extension has occurred transurethral resection or perineal prostatectomy offers relief from urinary retention, and high voltage therapy often relieves the pain from metastases.

CONCLUSION

In conclusion, may I repeat that only the hyperplastic or fibrous mass may be removed by suprapubic "prostatectomy" or by transurethral resection. To enable one actually to remove the prostate gland, a perineal exposure is necessary. Only by perineum can the posterior lobe (the site of 75 per cent of malignant neoplasms of the prostate) be removed together with the compressed remnants of the functioning prostatic glands. Only by maintaining a clear conception of the various pathologic lesions which may involve the prostate and after proper study should one decide on the rational surgical procedure in each case. To treat all types of surgical prostatic disease by one method is not sound from a pathologic viewpoint. Surgeons are rightly considered inadequate if through lack of preliminary investigation, study and understanding of the pathologic process present they fail to approach the problem by the method particularly fitted to give adequate permanent relief.

SUMMARY

1. Each of the three main types of prostatic operation has its place, yet none should be used to the exclusion of the others.

2. The origin, extent and location of prostatic disease should always be considered in selecting the proper surgical approach to relieve the patient completely and permanently from his prostatic disorder.

3. Prostatic calculi may be more completely removed by subtotal perineal prostatectomy, with less morbidity and more permanent cure.

4. Perineal exposure, with removal of the posterior lobe of the prostate, is the only method of adequately removing an early malignant neoplasm of this portion of the prostate.

5. Newer perineal prostatic operations permit adequate control of hemorrhage and offer a means of completely removing the diseased prostate, often preventing postoperative morbidity, with greater assurance of permanent lasting relief from prostatism.

901 Lexington Avenue.

ABSTRACT OF DISCUSSION

Dr. Hermon C. Bumpus Jr., Pasadena, Calif.: Many years ago Dr. Geraghty reviewed a series of cases of cancer of the prostate and found the posterior lobe to be the most frequent site of malignancy. Not so long ago, Kahler examined by serial section over 400 glands and found the lateral lobes more frequently involved than the posterior lobes. The gist of Dr. Henline's paper is that, as every fifth patient with prostatism presumably has a malignant condition present, one is not justified in doing other than a perineal prostatectomy for fear the malignant condition may invade the posterior lobe. Perineal surgery is a highly technical procedure taught in relatively few clinics. How can the average surgeon expect to master this technic and at the same time acquire the more involved and difficult one of transurethral resection? However, all who have been trained in the fundamentals of surgery can do a suprapubic prostatectomy. This might seem deplorable if it did in fact deprive every fifth prostatic patient of a cure of his disease and statistics showed that perineal prostatectomy actually cured any appreciable number of cases. Such, unfortunately, is not the case, owing to the fact that the perineal lymphatics of the prostate drain directly into the sacrum. Careful pathologic investigation has shown that there is no relationship between the extent of the malignant growth and the involvement of these perineal lymphatics. As a result, the early discovery of this disease means little as far as the chances of its cure are concerned but a great deal as far as the duration of the disease is concerned, for there are numerous untreated patients, except for the relief of their obstruction, who have lived over a decade from the time of its discovery. It is to be regretted that our literature does not contain reports of any series of patients treated by the perineal prostatectomy in which a sufficient number lived even over a five year period to make one certain that their survival was not due as much to the relief of their obstruction and the natural slow progress of the disease as to the eradication of the malignant growth. With few exceptions, urologists are far from satisfied with their end results in the treatment of prostatic cancer. In the great majority of cases relief of obstruction is what prolongs life, and our final results depend on the completeness with which this is done.

Dr. Charles C. Higgins, Cleveland: Dr. Henline's initial sentences are of prime importance. Surgical diseases of the prostate present so many variations that any one surgical procedure will fail to obtain the best results in all cases. Patients enter the hospital with acute obstruction who cannot have a catheter introduced into the bladder, and a suprapubic puncture or cystotomy must be done. Some of these patients can have the resectoscope introduced after preliminary drainage, but others cannot. Another group of patients are those with complete retention in which futile attempts to catheterize and evacuate the urine have been made. Metal sounds have been passed, the urethra has been torn, the penis is swollen, and the patient may have urethritis or unilateral or bilateral epididymitis. In these instances I would hesitate to perform a transurethral resection. A third group are patients with carcinoma of the prostate. I agree with Dr. Bumpus in that, when it is impossible to remove the malignant disease surgically in a very large prostate, transurethral resection is preferred. The fact must be appreciated that at some time the malignant lesion in the prostate was a local disease. Therefore, if a small nodule is palpated in a man aged 50, regardless of whether or not he has residual urine, one is justified in exposing the prostate by the perineal approach, securing a biopsy. If it is not malignant and merely an adenoma, the incision can be closed; if it is malignant, a radical procedure can be undertaken. The morbidity is much less in suprapubic prostatectomy, but personal preference also influences the choice of operation. The patients who present some discussion are those with grade 3 and grade 4 enlargements of the prostate. A sufficient amount of prostatic tissue can be removed to provide the patient an adequate channel so that he can entirely empty his bladder. However, many of these patients have a persistent pyuria which does not clear up until the prostate is removed. Microscopic examination of this prostatic tissue shows numerous infarcts and multiple abscesses. In the majority of instances it is not necessary to have an intensive preoperative regimen for patients who undergo transurethral surgery, provided there are no complications. However, the complications that may occur following operation must be anticipated. I have seen postoperative hemorrhage after transurethral resection. The patient loses large amounts of blood, which necessitates taking the patient to the operating room, evacuating the clots from the bladder and again introducing the resectoscope to control the bleeding point.. Occasionally, postoperative pneumonia will develop. If that patient's preoperative routine has been minimized, a higher operative mortality may be anticipated.

Dr. Franklin Farman, Los Angeles: I should like to present a somewhat different approach to the problem of prostatic surgery. Most indications for surgery are based on the anatomicopathologic picture found at the bladder neck; however, as a rule only the doctor himself understands this setup. In talking to patients I have found the following outline useful; most people place themselves in certain age groups and likewise does the "prostatic": From 50 to 60, resection or endocrine therapy; from 60 to 70, resection or one stage prostatectomy; from 70 to 80, two stage prostatectomy or resection; from 80 to 90, fractional resection, delayed two stage, permanent cystostomy, "catheter life." Between the ages of 50 and 60 one

encounters more frequently early benign adenoma, fibrous bar or commissural hypertrophy alone, and for these patients resection is ideally suited; mortality is nil and the functional result is good. At this stage, endocrine treatment (testosterone) is being tried with moderately encouraging results, and it is my belief that the field for glandular replacement therapy is greatest in the incipient stages of prostatic disease. Between the ages of 60 and 70, more extensive benign prostate enlargements and early carcinoma make their appearance. Life expectancy is fully ten years, and any contemplated surgery should keep this in mind, so as to insure the patient complete relief thereafter. Does resection always do this? During the past winter I have seen 5 patients on whom resection has been performed by five different doctors in the Midwest, all with recurrence of bladder difficulty, hematuria, stricture or infected residual urine. As the later decades of life are reached, complications are more frequent, operative hazard is increased and differentiation of cases becomes more necessary. In the age group 70 to 80, grade 3 and 4 benign adenoma, frank carcinoma, acute and long standing retention, urinary infection and lithiasis, damaged kidney function, arteriosclerosis, myocarditis and hypertension are factors of greater or lesser significance. Life expectancy is correspondingly shortened. The choice of method here has great influence on mortality and morbidity and depends on personal skill, available hospital equipment and assistance, the locality in which the patient resides and the objective in treatment—relief of symptoms or cure. Although the mortality rate is somewhat higher for total prostatectomy than for resection in this group, morbidity figures generally are lower. Paradoxical as it sounds, resection is advocated by some for both the "good risk" and the "poor risk" patient. This is true to a certain extent when considering the last age group beyond 80 years. Life expectancy is short and palliative relief usually is the objective, which may be accomplished by partial or fractional resection, delayed two stage, or permanent cystostomy.

DR. V. D. LESPINASSE, Chicago: Dr. Henline's paper is a plea to consider prostatic disease as a group of diseases, to consider it the same as we do intracranial, abdominal or pleural disease. There are many methods available and there are many different types of pathologic conditions that we need to relieve. Our methods of examination give us three aspects: (1) the intravesical aspect, (2) the perineal and (3) the urethral. Those methods will demonstrate the exact pathologic condition, and the problem is how best to get rid of that condition. If it is a small obstruction in the urethra there is no advantage, in fact it is foolish to do anything but a local urethral destruction. If there is a pathologic lesion in the bladder, it seems to me that the easiest and best way to remove it is to go into the bladder. If the pathologic process is on the rectal side of the prostate, then the most accessible way to get at it is through the perineum. If one studies the case better and considers the pathologic condition of the particular patient, one most certainly will obtain better results. As far as the various points of technic are concerned and the saying of carte blanche to do it above or below, I don't think that is profitable to discuss: For my own abilities I can do a total anatomic removal of the prostate suprapubically from the membranous urethra to the ureteral orifices, unite the trigon of the bladder to the urethral orifice as it comes through the urogenital ligament, and wrap a few Wilson's muscles or the puboprostate ligament around it and obtain a nice result with little mortality. Patients are well pleased and their carcinoma is no longer in their body. Whatever way one works, one must pay attention to the pathologic lesion and remove it; then one will have some excellent results.

DR. ROY B. HENLINE, New York: Dr. Bumpus mentioned the pertinent fact that pathologists often disagree on the diagnosis of prostatic cancer. Various articles written on this subject tend to confuse the issue on the origin and progression of prostatic cancer. In my text I did not refer to Dr. Geraghty's original paper on prostatic cancer when I stated that approximately 75 per cent of these cancers originated in the posterior lobe of the prostate. I referred to two recent, very careful, investigations by Moore and Rich, who independently found the posterior lobe of the prostate to be the site of origin of approximately 75 per cent of prostatic cancers. As long as pathologists agree that most prostatic cancers originate in the posterior lobe, we as urologists should treat our patients by that method which will adequately remove this involved area. Apparently Dr. Bumpus misunderstood one of my purposes in presenting this paper. For fear that others may have misunderstood I should like to reiterate that I am not recommending perineal prostatectomy to the exclusion of other types of surgery. The latest statistics show that perineal prostatectomies are performed in approximately 30 per cent of patients, transurethral resection in 60 per cent and suprapubic enucleation in 10 per cent. I believe that the type of surgery employed should depend on the pathologic condition present in the prostate. To attain this goal, perineal prostatectomy must be employed for some patients. Dr. Bumpus also stated that the technic of perineal surgery is difficult. I find it more difficult to teach the resident staff transurethral resection than perineal surgery. I believe that proper transurethral surgery is among the most difficult urologic procedures one has to perform. If Dr. Bumpus had done as many perineal prostatectomies as he has transurethral resections he would find that learning perineal surgery is much easier than learning transurethral surgery. If I have brought out no other point, I should like once more to stress that not all patients with prostatic disease should be treated by any one method.

INTRASPINAL THERAPY OF NEUROSYPHILIS

ROBERT R. KIERLAND, M.D.
AND
PAUL A. O'LEARY, M.D.
ROCHESTER, MINN.

A great number of articles in the literature from 1910 until the early 1930's attest the value of intraspinal methods in the treatment of neurosyphilis. However, since the advent and ensuing favor of various other measures, this method of therapy gradually has been discarded by many syphilotherapists and we think unjustifiably so.

In appraisal of the methods of treatment dealt with in three papers from the Cooperative Clinical Group,[27] it must be remembered that the various forms of supplemental therapy in general were used only after routine therapy had failed and many of the patients had more than one type of supplemental treatment. The conclusions that were reached in the aforementioned papers, therefore, do not constitute a true comparative evaluation of the symptoms of treatment but rather constitute contrast of one method with another when simpler methods failed to produce the desired clinical and serologic results. For example, a comparison of intraspinal therapy with routine treatment cannot be made on a numerical basis because intraspinal therapy was administered usually to patients whose condition was resistant and who had failed to improve under routine treatment. However, the three reports clearly indicate the value of intraspinal therapy in certain forms of neurosyphilis, especially in mild or early tabes dorsalis and in the asymptomatic, meningeal and meningovascular forms of neurosyphilis.

From the Section on Dermatology and Syphilology, Mayo Clinic.

Read before the Section on Dermatology and Syphilology at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 5, 1941.

Owing to lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the author's reprints.

27. O'Leary, P. A.; Cole, H. N.; Moore, J. E.; Stokes, J. H.; Wile, U. J.; Farran, Thomas; Vonderlehr, R. A., and Usilton, Lida J.: Cooperative Clinical Studies in the Treatment of Syphilis: Asymptomatic Neurosyphilis, Ven. Dis. Inform. **18**: 45-65 (March) 1937; Tabes Dorsalis, ibid. **19**: 367-396 (Nov.) 1938. O'Leary, P. A.; Kierland, R. R.; Cole, H. N.; Binkley, G. W.; Wile, U. J.; Stokes, J. H.; Ingraham, N. R., Jr.; Moore, J. E.; Padget, Paul; Kemp, J. E.; Farran, Thomas; Vonderlehr, R. A.; Usilton, Lida J., and Vandoren, Eleanor: Cooperative Clinical Studies in the Treatment of Syphilis: Asympomatic Neurosyphilis, ibid., to be published.

THEORIES OF THE MECHANISM OF ACTION OF INTRASPINAL THERAPY

There are essentially four theories concerning the mechanism of action of the various forms of intraspinal therapy. These are that:

1. The amount of arsenic introduced into contact with the cerebrospinal fluid, although small, has a definite antisyphilitic action.

2. As a result of the introduction of the foreign material there results an increased permeability of the meninges due to meningeal irritation, which increases the concentration in the cerebrospinal fluid of arsenic, bismuth and other antisyphilitic drugs administered intravenously or intramuscularly.

3. The antibodies which are brought forth in the blood as the result of the primary injection of arsphenamine are transferred in the serum to the cerebrospinal fluid.

4. There is a nonspecific protein effect produced by the introduction of the blood serum into the spinal canal.

It is probable that all these theories play more or less definite roles in the production of the good clinical and serologic results obtained from intraspinal therapy. However, it is not our intention to discuss in detail these theoretical considerations of the mechanism of action of intraspinal treatment or to evaluate other similar types of treatment, such as spinal drainage and cisternal drainage, although we have had some experience with such methods. The results to be given in this report deal only with the so-called Swift-Ellis type of intraspinal therapy. For several years, reinforcement of the blood serum with minute doses of arsphenamine according to the method of Ogilvie was carried out but was discontinued because the results did not warrant the additional hazard incurred by the method.

SCOPE OF THE STUDY

This survey is based on the study of 370 patients suffering from various types of neurosyphilis who had received a total of two thousand, three hundred and sixty-nine intraspinal treatments, an average of six and four-tenths treatments per patient. The largest number of treatments administered to one person was thirty-nine and the smallest number one. These patients were treated during the years 1924 through 1927 and were selected because of the opportunity thus presented for a prolonged follow-up study.

TECHNIC OF INTRASPINAL THERAPY

Twenty to thirty minutes after an intravenous injection of arsphenamine or neoarsphenamine, 50 cc. of blood is aseptically withdrawn into sterile containers. The blood is allowed to stand until the serum separates and the serum then is pipetted off into another sterile container of a capacity of approximately 50 cc. The remaining clotted blood and serum are centrifuged and the remaining serum is again pipetted. Usually, 20 to 25 cc. of serum is obtained. The serum is then stored in a refrigerator overnight and in the morning it is heated to 56 C. for seventy minutes to destroy the complement; it is then centrifuged again to separate the few erythrocytes that may be present. This must all be done by means of a rigidly strict sterile technic.

Lumbar puncture is then done in the usual manner (in the fourth or fifth lumbar interspace) and 20 to 25 cc. of cerebrospinal fluid is removed, of which 10 cc. is saved for the globulin, cell count, protein, colloidal gold and serologic determinations; the rest of the fluid is discarded. The blood serum is then added to a gravity tube and allowed to flow into the subdural space by gravity; no pressure or force of any type need be employed when the serum is being introduced into the spinal canal. The patient is then kept in bed for twenty-four hours. The foot of the bed is elevated about 8 inches for six to eight hours and the patient is urged not to use a pillow for twelve to eighteen hours.

Each intraspinal treatment is administered at two week intervals and three such treatments are considered to constitute a course. During this six week period the neurosyphilitic patient also receives routine treatment in the form of an injection of arsphenamine once weekly, heavy metal (either mercury succinimide five times weekly or bismuth two or three times weekly) and potassium iodide by mouth or sodium iodide intravenously.

The patient who has cardiovascular syphilis and only coincidental neurosyphilis for whom the administration of arsphenamine is contraindicated may receive so-called foreign arsphenaminized serum with benefit. In this modification the arsphenamine is administered first to another syphilitic patient; blood is withdrawn from this patient and the serum is extracted in the usual manner. The arsphenaminized serum thus obtained is then administered intraspinally to the patient who has both cardiovascular syphilis and neurosyphilis.

REACTIONS

In the total number of two thousand three hundred and sixty-nine treatments there were two hundred and twenty-nine reactions in 109 patients of sufficient degree to warrant their having been recorded in clinic files, an incidence of 9.7 per cent. These reactions were classed as "slight," "moderate" or "severe";[28] there were ninety-three slight reactions (3.9 per cent of the total number of treatments), ninety moderate reactions (3.8 per cent) and forty-six so-called severe reactions (1.9 per cent).

It is interesting to note that 39 per cent of all reactions occurred in a small group of 18 patients, and in this group it was necessary to discontinue intraspinal therapy for only 1 patient, a tabetic patient in whom increasing ataxia developed after intraspinal treatment; and, although there was some question as to the part played by the treatment in the production of the ataxia, it nevertheless was included among the reactions.

Reactions occurred most frequently among the tabetic group of patients for whom therapy provoked increased pains in the leg and/or gastric crises of varying degrees. There were 8 patients among the entire group for whom it was necessary to discontinue intraspinal therapy because of the occurrence of reactions of one type or another.

The reactions noted most commonly were those referable to ordinary lumbar puncture and slight to severe headache, which may or may not have been associated with nausea and vomiting. Two hundred of two hundred and twenty-nine reactions constituted headache, nausea and vomiting and increased pains in the legs.

Among the severe complications was 1 instance of meningitis, from which the patient recovered within three weeks' time without residual difficulty. In 5 cases complications developed which were referable to the

28. Slight reaction: Symptoms of the type of headache which often follows lumbar puncture, increased pains in legs and mild degrees of nausea and vomiting lasting less than twenty-four hours. Moderate reaction: Reactions in this group sometimes endured up to forty-eight hours; patients sometimes required periods of rest in bed and sedation. Symptoms were of the same general type as those which follow lumbar puncture. Severe reaction: Reactions of the severe type lasted for more than forty-eight hours and required rest in bed and sedation. Provocation of pains in legs and gastric crises by the Swift-Ellis treatment and the occurrence of meningism also are included in this group.

bladder. Three of the patients who had vesical complication received additional intraspinal treatment without the recurrence of complications. For the other 2 patients additional Swift-Ellis treatment was not attempted. In 2 cases transverse myelitis developed after the use of intraspinal therapy; however, both of these patients recovered without evidence of residual neurologic effects and 1 was able to tolerate additional intraspinal therapy without subsequent disturbance. One patient manifested saddle anesthesia; however, he also was able to tolerate Swift-Ellis treatment later without any untoward reactions.

It is to be remembered that, during the twenty-four year period in which intraspinal therapy has been used in the clinic, more than twelve thousand such treatments have been administered and that complications such as meningitis, retention of urine and transverse myelitis have developed among patients other than those considered in this study. Such reactions, however, are uncommon, since in the present series of two thousand three hundred and sixty-nine consecutive intraspinal treatments administered during the years 1924 to 1927 only nine troublesome complications occurred.

RESULTS

The consecutively selected records in this study concern a group of 370 neurosyphilitic patients who received one to thirty-nine intraspinal treatments in conjunction with therapy with arsphenamine, neoarsphenamine or sulfarsphenamine and mercury or bismuth compounds and, in the majority of instances, with iodides. Two hundred of these patients were observed and/or treated for five years or longer. These 200 patients who have had treatment and have been observed for periods of five years or more form the basis of the clinical and serologic results to be described hereafter. For 178 of these 200 patients, treatment was started by a combination of routine and intraspinal therapy.

The various manifestations of neurosyphilis in this group were diagnosed as follows: asymptomatic, tabes dorsalis, the tabetic form of dementia paralytica, dementia paralytica, meningeal and meningovascular. There were only 2 cases of purely vascular neurosyphilis, and these have been included with the meningovascular group of cases of neurosyphilis and the tabetic form of dementia paralytica.

Asymptomatic Neurosyphilis.—Thirty-two patients (16 men and 16 women) had asymptomatic neurosyphilis; their ages varied from 5 to 50 years. Nine of these patients previously had received various degrees of intravenous and intramuscular antisyphilitic therapy [29] and in 4 of the 9 the reaction to serologic tests of the blood was negative when they came under our observation. With routine treatment, plus the addition of intraspinal therapy, the blood became normal for 24 additional patients. According to classification by types of cerebrospinal fluid encountered, 7 patients had group I spinal fluid, 22 had group II spinal fluid and 3 had group III spinal fluid. Explanation of the classification of the three types of spinal fluid may be found in table 1. Twenty-five patients obtained reversal of the condition of the spinal fluid by means of intraspinal and routine treatment alone. Reactions to tests of the spinal fluid of 4 patients remained positive in spite of treatment, and in 3 patients reactions to such tests relapsed to positive.

Four patients received malaria therapy in addition to routine and intraspinal treatment. One of these 4 patients, because of previous intensive treatment before the patient was seen by us, received therapy with malaria fever first, and then routine and intraspinal treatment. The spinal fluid of this patient first became normal nine years after fever therapy. The second patient received fever therapy with malaria because the reactions of tests of the spinal fluid relapsed to positive, whereas the other 2 patients of this group of 4 received treatment with malaria because of persistently positive reactions to tests of the spinal fluid. Reactions to such tests in 1 of these 2 cases remained positive five years after the addition of malaria therapy, whereas the other patient had negative reactions to the tests. One other patient received sodium N-phenylglycinamide-p-arsonate (tryparsamide) without benefit to the spinal fluid.

There were 3 cases in which reactions to tests of the spinal fluid relapsed and became negative with additional treatment. Reactions to tests of the fluid of 1 of the patients relapsed on routine treatment, and the spinal fluid became normal with the addition of intraspinal treatment. The second patient required therapy with malaria to bring about the desired normality of the spinal fluid, whereas the spinal fluid of the third patient reverted to normal with further intraspinal treatment and remained normal.

TABLE 1.—*General Classification by Group of Spinal Fluid According to the Degree of Abnormality Found on Examination by Each of the Four Indicated Procedures*

Group	Reaction to Test Indicated: Wassermann	Reaction to Test Indicated: Globulin	Cells per Cu. Mm.	Colloidal Benzoin or Gold Curve
I	Negative or weakly positive	Positive or negative	10-20	000 000 333 000 000 0 0 0 3 3 3 3 0 0 0
II	Positive	Positive	20-100	000 000 333 000 000 0 0 0 3 3 3 0 0 0 0 021 221 333 310 000 0 1 2 5 5 5 3 0 0 0
III	Strongly positive	Positive	30-100 10-30* 10-30†	333 333 321 000 000 5 5 5 5 5 4 3 2 1 0

* Large lymphocytes.
† Polymorphonuclear cells.

In 17 of 28 cases reactions to tests of the cerebrospinal fluid that became negative did so within a year or less; reactions in 6 more cases became negative within one to two years, whereas in the other 5 cases the reactions became negative within two to nine years.

The 32 patients who had asymptomatic neurosyphilis received from one to thirteen intraspinal treatments each, in addition to arsphenamine, a heavy metal and iodides; of these 32 patients, 29 received three or more intraspinal treatments in conjunction with routine treatment. These 29 patients underwent a total of two hundred and thirty-eight intraspinal treatments, an average of eight per patient.

Symptomatic neurosyphilis did not afflict any patient in the group.

Tabes Dorsalis.—Fifty of 200 neurosyphilitic patients had tabes dorsalis; 6 of these 50 had optic atrophy. The youngest was 8 years of age and the oldest was 55. All but 7 were males. Twenty-one patients had had some type of antisyphilitic treatment before coming under our care. Results of serologic tests of the blood

29. Hereinafter when we speak of "previous therapy" we will mean "antisyphilitic treatment administered prior to the patient's coming under our care." Almost invariably this treatment was inadequate, according to present day standards; that is, it had consisted of less than thirty intravenous injections of arsphenamine and sixty intramuscular injections of a heavy metal. Many of these patients had previously received only oral medication with mercury or iodides.

of 8 patients who had received no previous treatment were negative when the patients were first seen, and results of such tests of 6 patients who had received previous antisyphilitic treatment were negative when the patients were first seen. At the end of our treatment and observation period, reactions to serologic tests of the blood of 36 patients were negative, whereas such reactions concerning 14 patients were still positive.

Seven persons had group III spinal fluid, 33 had group II fluid and 10 had group I fluid. By virtue of the use of all types of treatment at our command, at the end of treatment and observation reactions to tests of the spinal fluid of 40 patients became negative, whereas such reactions among 10 patients were still positive (in all the fluid was of group I). Thirty patients received routine and intraspinal treatment alone; the spinal fluids of 26 patients became normal and reactions to tests of the spinal fluid of 4 patients remained positive. Condition of the spinal fluid relapsed in 5 instances, but in each of these instances the spinal

TABLE 2.—*Clinical Results of Intraspinal and Routine Treatment Before Institution of Other Types of Treatment*

Type of Neurosyphilis Present	Cases	Result: Decidedly Improved No.	%	Slightly Improved No.	%	Arrest or No Change No.	%	Condition Progressed No.	%
Asymptomatic	31	..	....	..	....	..	....	0	....
Tabes dorsalis	48	28	58.3	13	27.0	5	10.4	2	4.2
Tabetic dementia paralytica, vascular and meningovascular	45	25	55.5	9	20.0	6	13.3	5	11.1
Dementia paralytica	16	5	31.2	3	18.7	6	37.5	2	12.5
Meningeal	38	34	89.5	2	5.3	2	5.3	..	....

fluid became normal with further treatment of the patients, 3 of whom received malaria therapy and 2 of whom received Swift-Ellis treatment only.

Of the 19 patients who received malaria treatment plus intraspinal therapy, 5 still had spinal fluid which reacted positively to tests.

Only 2 patients in this particular group received tryparsamide; 1 received it alone with intraspinal treatment and 1 received it with both malaria therapy and intraspinal treatment. Nineteen patients received malaria treatment, 3 because of relapse of reactions to serologic tests, 2 because of clinical progression of the disease, 1 because of reactions from intraspinal treatment, 6 because the condition of the spinal fluid failed to respond and 5 because intraspinal treatment gave neither clinical progression nor improvement. For 2 of these 19 patients treatment had been started with fever produced by means of malaria. In the 2 cases in which clinical progression of the disease occurred, the condition in both became clinically arrested with the addition of malaria therapy. Of the 5 patients who did not respond to treatment, 3 remained in the same condition, 1 improved remarkably and 1 improved slightly.

In the group of 30 patients who received routine treatment plus intraspinal treatment alone there was no progression of symptoms, subjectively or objectively, whereas 24 of these patients obtained decided improvement;[30] 4 obtained slight improvements and 2 exhibited no change.

30. This is considered as constituting subjective improvement manifested by the patient and expressed by him and noted by improvement in somatic complaints, gain of weight, improved appearance and sense of well being. Neurologic signs (objective symptoms), such as pupillary and other changes in reflexes, sense of position and pain, did not change with the patients under antisyphilitic treatment and observation.

However, when intraspinal treatment in the whole group of 50 patients failed to produce satisfactory clinical results, the use of other types of supplementary treatment such as malaria therapy and/or tryparsamide produced for 31 patients decided improvement in subjective signs and symptoms, slight improvement in such signs for 13 and no improvement for 6. The condition of no patient progressed.

Six of the 50 patients had optic atrophy in addition to other signs and symptoms of tabes dorsalis. For 2 of these 6 patients the type of antisyphilitic treatment first used was malaria therapy; 2 of the 6 showed progression of optic atrophy under intraspinal treatment and received malaria therapy in an effort to halt this progression. Two of the 6 patients had no further loss of vision on receiving routine and intraspinal therapy.

For only 4 tabetic patients did the spinal fluid become normal in less than a year. The longest period required for a positive reaction to revert to negative was eight years; this period was required for 3 patients. In the majority of cases the reactions to tests of the fluid became negative in one year to three years.

One patient received less than three intraspinal treatments. The remaining 49 patients suffering from tabes dorsalis received a total of three hundred and ninety-one such treatments, an average of about eight per patient.

Dementia Paralytica.—Thirty-four of these 200 neurosyphilitic patients had dementia paralytica, and of these 34, 13 had received previous antisyphilitic treatment. Generally dementia paralytica encountered among these patients was in a very early stage, and although many of them were euphoric and had grandiose ideas, in none was the condition in a vegetative stage. The youngest patient was 10 years old and the oldest was 53 years old. There were 2 women in this group. One patient had not received previous treatment and was found to have negative reactions to serologic tests of the blood. In 1 case reactions to serologic tests of the blood were negative, the patient having had previous treatment. Eleven patients still had positive reactions to serologic tests of the blood after treatment and an observation period of five years or longer.

Twenty-five patients had cerebrospinal fluid of group III, 8 had fluid of group II and 1 had fluid of group I on admission; at the end of our treatment and observation, 23 had normal spinal fluid, 9 had fluid of group I and 2 still had fluid of group III.

Among the 23 cases in which reactions to tests of the spinal fluid reverted to negative, in only 1 did the reaction revert to negative in less than a year; the spinal fluid of 2 persons became normal in one to two years; in 15 cases from two to five years were required for the reactions to reverse, and in 5 cases five years or more were required for the reactions to become negative. The longest period required for the fluid to become normal was thirteen years.

In 4 cases the reactions to tests of the spinal fluid relapsed; in 3 cases the reactions reverted to negative with further treatment of the patients, and in these three cases fever treatment with malaria was administered because of the relapse.

It is difficult to appraise the value of intraspinal therapy in this particular group of patients because of the fact that 16 patients also received malaria therapy and intraspinal treatment, 1 received treatment with tryparsamide and intraspinal therapy and 14 received therapy with both malaria and tryparsamide in addition to intraspinal treatment. Only 3 patients received intraspinal treatment and routine treatment alone, of whom

2 were decidedly improved and one was slightly improved. Thirteen others received the Swift-Ellis treatment before the institution of other methods of treatment. Three of these 13 patients received other types of therapy because of relapse of the reactions to tests of the spinal fluid; 10 of these 13 patients received other therapy because of failure of improvement as manifested by either reactions to serologic tests or remission of clinical symptoms. In 2 cases the disease showed evidence of clinical progression on intraspinal treatment of the patients but improvement was obtained with malaria therapy.

For 17 patients the first treatment administered was malaria, and 1 received tryparsamide. Follow-up therapy consisted of intraspinal treatment or administration of tryparsamide, or both. Of 11 who received malaria therapy first, plus intraspinal and routine treatment alone, 8 were decidedly improved, 2 were slightly improved and 1 became worse. Of 14 patients who received malaria therapy, followed by intraspinal treatment plus therapy with tryparsamide, 8 patients were decidedly improved, 4 were slightly improved and 2 became worse.

With all types of treatment, 22 patients were decidedly improved, 9 were slightly improved and 3 became worse.

Two patients received only one intraspinal treatment each; the largest number of treatments received by any 1 patient was thirty-nine. A total of three hundred and fourteen injections were made for 32 patients, an average of nine and eight-tenths injections each.

Meningeal Neurosyphilis.—Thirty-nine patients out of the series of 200 had meningeal neurosyphilis; the youngest of these was 21 years old and the oldest was 60 years old. All but 5 were men. Twelve had received previous antisyphilitic treatment before they came under our care. Two patients who had negative reactions to serologic tests of the blood had received no previous treatment, and 1 patient who had negative reactions to serologic tests of the blood had received previous treatment. Under treatment, reactions to serologic tests of the blood became negative in all but 3 cases.

TABLE 3.—*Serologic Effects of Intraspinal and Routine Treatment on the Cerebrospinal Fluid Before Institution of Other Methods of Therapy*

Type of Neurosyphilis Present	Cases	Condition of Cerebrospinal Fluid (Reaction): Complete Reversal No.	%	Improved but Still Positive No.	%	Stationary or Worse No.	%	Relapse * No.	%
Asymptomatic....	31	25	80.6	3	9.7	1	3.2	3	9.7
Tabes dorsalis....	48	26	54.1	15	31.2	5	10.4	5	10.4
Tabetic dementia paralytica, vascular and meningovascular.....	45	23	51.1	13	28.8	6	13.3	8	17.7
Dementia paralytica.....	16	2	12.5	7	43.7	3	18.7	4	25.0
Meningeal.....	38	33	86.8	3	7.9	1	2.6	2	5.3

* Partial overlapping of percentages with those in column headed "complete reversal."

All reactions to tests of spinal fluid were positive. The fluid in 9 cases was of group III, in 1 it was of group I and in the remaining 29 cases it was of group II. In all but 3 of the cases reactions to tests of the spinal fluid became negative during the five year period of treatment and observation. In 2 of these 3 cases spinal fluid of group III was present and malaria therapy was administered without complete reversal of the condition of the spinal fluid; the other patient showed no improvement whatever in the condition of the spinal fluid, although results of serologic tests of the blood did become negative. The patient showed no progression of symptoms in an eight year treatment and observation period.

TABLE 4.—*Serologic and Clinical Results Obtained from Routine Treatment Only in Neurosyphilis: Statistics of Cooperative Clinical Group*

Type of Neurosyphilis Present	Reversal of Reactions to Serologic Tests of Cerebrospinal Fluid: Number, Total	Reversals, Number *	Per Cent *	Decided Clinical Improvement: Number, Total	Decided Improvement, Number	Per Cent
Asymptomatic.....	203	139	68.5			
Tabes dorsalis.....	396	114	28.8	286	44	15.4
Tabetic dementia paralytica, vascular and meningovascular.....	187	69	36.8	148	41	27.7
Dementia paralytica	57	6	10.5	25	4	16.0
Meningeal.....	88	50	56.8	71	55	77.5

* Total of cases in which reactions to tests were reversed at five years.

Of 36 instances in which the spinal fluid became normal, the fluid in 21 became normal within a year or less, in 8 instances the fluid became normal within the one to two year period and in the other 7 instances the fluid became normal in the two to five year period. In 2 instances reactions to tests of spinal fluid relapsed to positive; in both the reactions became negative again and remained so. One relapse occurred in an uncooperative patient, the other in a patient whose reactions had relapsed twice and who, at the second relapse, received malaria treatment. None of the patients received tryparsamide, but 8 received malaria therapy or typhoid vaccine. One patient received malaria therapy first of all because his spinal fluid was found to be of group III. All but 2 of the remaining 7 patients (of the 8 who received malaria therapy or vaccine) had obtained decided clinical improvement before we proceeded with fever therapy; the sixth patient showed improvement only after the addition of typhoid vaccine to the therapeutic armamentarium, whereas the seventh patient showed no improvement after any type of therapy; this particular patient had convulsions, and it is questionable how much of a part the syphilis had in the production of the condition. Intraspinal therapy used alone or administered before other methods of therapy accounted for thirty-four decided improvements and for two slight improvements. There were no clinical data in 1 case.

No patient in this group of 39 received less than three intraspinal treatments, and the most received by 1 person was thirteen. There was a total of three hundred and thirteen Swift-Ellis treatments, or an average of about eight treatments per patient.

Tabetic Dementia Paralytica; Vascular and Meningovascular Neurosyphilis.—In this series 19 had the tabetic form of dementia paralytica, 2 had vascular neurosyphilis and 24 had the meningovascular form of neurosyphilis, a total of 45 patients. The ages varied between 4 and 55 years, and only 8 of the 45 patients were women. Seven patients had negative reactions to serologic tests of the blood, and all but 2 of these 7 patients had undergone previous treatment. After

treatment and observation by us, all but 6 patients had negative reactions to serologic tests of the blood. Twenty of the 45 patients in this group had received previous antisyphilitic treatment.

In 15 cases the spinal fluid was classified as being of group III, 27 patients had spinal fluid of group II and 3 had spinal fluid of group I.

In 34 cases the spinal fluid became normal under treatment and observation; in the majority of these cases the fluid became normal within one and three years. In 23 cases the fluid became normal with the use of routine and intraspinal treatment alone, and in 11 cases the addition of malaria or tryparsamide therapy or both to the routine and intraspinal routine of treatment accomplished this result. In 11 cases reactions to tests of the spinal fluid remained positive; 8 of the 11 patients received fever treatment with malaria, 2 received therapy with tryparsamide and 1 patient received only routine and intraspinal treatment.

In 8 cases the condition of the spinal fluid relapsed; reactions in 7 of these cases reverted again to negative with further treatment, in 3 of these the reactions became negative when further intraspinal treatment alone was given, in 3 cases the reactions became negative with the addition of malaria treatment to the schedule of therapy, and in 1 instance of relapse after the use of both intraspinal treatment and malaria treatment, the reactions became negative with therapy with tryparsamide and typhoid vaccine.

Well defined clinical improvement was obtained for 18 patients who received routine and intraspinal treatment alone, whereas 4 patients showed slight improvement and 2 patients exhibited neither progression of symptoms nor improvement. Of 19 patients who received malaria therapy, 11 received it because intraspinal treatment had failed either clinically (6 patients) or serologically (5 patients); the other 8 patients, however, exhibited varying degrees of improvement before malaria therapy was employed (6 patients exhibited decided improvement and 2 improved slightly). Of the 2 patients who received intraspinal treatment plus tryparsamide, 1 showed much improvement and 1 slight improvement before tryparsamide was administered. With all types of treatment, 28 patients were decidedly improved, 11 were slightly improved, 5 showed no change and the condition of 1 patient progressed.

Only 1 patient received less than three intraspinal treatments, whereas the largest number administered to any patient was fifteen. The remaining 44 patients (of the 45 in this group) received a total of three hundred and eighty-one treatments, an average of eight and two-tenths Swift-Ellis treatments per patient.

Tables 2 and 3 show the results obtained clinically and serologically by intraspinal treatment before addition of the other therapeutic measures of malaria, typhoid vaccine and/or tryparsamide.

In table 4 the serologic and clinical results obtained from routine treatment only by the Cooperative Clinical Group are listed. These results should be compared with the results in our cases obtained by a combination of intraspinal and routine treatment, as set forth in tables 2 and 3.

COMMENT

A rather characteristic trend was noted of the condition of the cerebrospinal fluid of patients who subsequently exhibited improvement from intraspinal therapy. The first indication of benefit was found in the rapid diminution of the cell count, although not uncommonly after the first intraspinal treatment there was a slight increase in this count denoting meningeal irritation. With subsequent treatment, however, this increase was noted only occasionally. In many instances, especially in cases of meningeal, asymptomatic, meningovascular neurosyphilis and in tabes dorsalis, the cell count was within normal limits at the end of the first course of intraspinal treatment. Because of the lability and sensitivity of the cell count of the cerebrospinal fluid, a decrease of the cell count of the fluid is a helpful prognostic guide in forecasting the subsequent changes of the spinal fluid after intraspinal treatment.

Next in order of responsiveness to intraspinal treatment was the flattening of the colloidal gold or colloidal benzoin curves (table 1). This sign of improvement, however, frequently lagged behind improvement as denoted in the cell count for from months to several years, whereas changes signifying improvement in results of the globulin and Wassermann tests were the last to appear. The latter two factors closely paralleled each other.

The value of classification of observations concerning the spinal fluid into three groups was further substantiated by this study. Patients with fluid of group I or group II generally do well under intraspinal therapy, whereas those who have fluid of group III have a tendency to be resistant to all but the combination of nonspecific and specific remedies. Dementia paralytica, which is accompanied by the highest percentage of cerebrospinal fluid of group III, is resistant to Swift-Ellis therapy. However, as brought out by the Cooperative Clinical Group, meningeal neurosyphilitic persons who have fluid of group III respond more readily than do patients with dementia paralytica who have a similar type of cerebrospinal fluid.

In general, the shorter the duration of neurosyphilitic symptoms the more rapid the response to intraspinal therapy was found to be. This trend did not necessarily pertain to the duration of syphilis, because those patients among whom neurosyphilitic symptoms developed early in the course of their disease often were resistant to all forms of treatment.

Certain valid criticisms may be made of a paper of the type of this one. First is the fact that an adequate series of control cases is lacking. The ideal method would be to start routine treatment alone for a large number of patients suffering from neurosyphilis and then to begin routine treatment plus one type of supplemental therapy for another similar group of patients for purposes of subsequent statistical study. It was obviously impossible to utilize such a procedure for a retrospective survey such as this one. Another criticism which may be brought forth by the opponents of intraspinal therapy is the assumption that other methods such as spinal drainage alone, or the introduction of unarsphenaminized serum, do as much good as the therapy we have described. Our answer to this is that in our experience such methods of treatment produced results, both clinical and serologic, which were inferior to those obtained with the Swift-Ellis technic.

In the present paper a selected group of neurosyphilitic patients were considered, namely those who were physically able to undergo intraspinal treatment because signs and symptoms of advanced phases of the disease had not been produced. In other words, intra-

spinal therapy was confined to patients who exhibited the early clinical signs of involvement of the central nervous system.

The fact that we have chosen patients who underwent treatment and observation for periods of five years or longer tends automatically to exclude many of the poor results obtained for patients with dementia paralytica and patients with the tabetic form of dementia paralytica, since these particular patients frequently die within five years after the onset of symptoms; on the other hand, many of the good results obtained for patients who had asymptomatic or meningeal neurosyphilis are excluded, since such patients disappeared from observation within this period.

The best results of intraspinal therapy were obtained for those neurosyphilitic patients who had the meningeal, asymptomatic and early tabetic manifestations of the disease. Patients who had early tabes dorsalis did decidedly better than those who exhibited signs of the advanced disease.

The poorest results were obtained for patients who had dementia paralytica. In the presence of this condition the main value of intraspinal treatment lies in postmalaria therapy if pronounced mental deterioration is not present. Intraspinal therapy is of less value than fever therapy and tryparsamide in the treatment of dementia paralytica.

The Swift-Ellis technic of intraspinal therapy in the management of some of the early manifestations of neurosyphilis is safe and the benefits gained from it by the patient far outweigh the incidence of untoward reactions to the procedure. In the present series there were only nine disturbing or potentially serious reactions in more than two thousand three hundred intraspinal treatments, and among none of these patients did permanent sequelae develop. The majority of these reactions were encountered by patients who had tabes dorsalis with evidence of involvement of the lower part of the spinal cord.

It is our opinion that treatment with malaria is more beneficial than Swift-Ellis therapy in producing arrest of the advancement of optic atrophy.

CONCLUSIONS

The indications for intraspinal (Swift-Ellis) treatment, provided the patient is in good physical condition and can tolerate the arsphenamines, may be summarized briefly as follows:

1. It is particularly of benefit for patients who have asymptomatic and meningeal neurosyphilis and for those who have early tabes dorsalis.

2. Intraspinal therapy of the Swift-Ellis type is of special value for patients who have the early manifestations of neurosyphilis when a trial with routine treatment has failed to produce clinical or serologic improvement.

3. The Swift-Ellis procedure is valuable postmalarial therapy in certain forms of neurosyphilis in which mental changes are not present.

4. When this procedure is employed, so-called foreign arsphenaminized serum may be used in cases of syphilitic cardiovascular disease with complicating neurosyphilis.

5. When the Swift-Ellis procedure is employed, a minimum of seven intraspinal treatments combined with routine therapy is desirable, although many patients do well with fewer treatments.

Contraindications to intraspinal therapy include, besides those common to the use of arsphenamine, the following: (1) patients who exhibit the clinical signs of neurosyphilis which persist in spite of adequate treatment in whom reactions to tests of the cerebrospinal fluid have become negative, (2) patients physically unfit, such as those who have advanced tabes dorsalis, (3) the presence of the signs and symptoms of advanced involvement and degeneration of the lower part of the spinal cord and (4) cases in which reactions from the treatment are more severe than those resulting from ordinary lumbar puncture.

Excellent serologic and clinical results may be obtained with treatment by a combination of intraspinal and routine therapy for patients who have certain forms of neurosyphilis. However, it is our practice to use other forms of supplemental treatment, such as fever therapy and/or tryparsamide, when satisfactory results fail to appear after a trial with intraspinal therapy.

ABSTRACT OF DISCUSSION

DR. HAROLD N. COLE, Cleveland: Drs. Kierland and O'Leary have brought out a fact that may not be well known, namely that it was Marinesco who in 1910 and 1911 first injected the serum of syphilitic patients into the same patients intradurally. Much has been written on the subject, though particularly in a period up to fifteen years ago. I wonder whether this has any significance. The writers' material is based on 2,369 intraspinal treatments given to 370 patients through the years 1924 to 1927, thus allowing prolonged follow-up study. There were reactions after 229 treatments, an incidence of 9.7 per cent, but the reaction after 200 of the 229 consisted of headache, nausea and vomiting and increased pain in the legs. The severe reactions were found more in persons with tabes dorsalis. In 5 of these they involved the bladder. In 2 transitory transverse myelitis developed, though 1 of them subsequently received further treatment without disturbance. In 1 there was meningitis, with recovery within three weeks. The results of therapy are limited to 200 patients observed for five years or more. The authors divide the evidence of neurosyphilis in the cases into asymptomatic manifestations, tabes dorsalis, the tabetic form of dementia paralytica, dementia paralytica and meningeal and meningovascular manifestations. Best results were achieved in cases of early involvement of the central nervous system—in cases of asymptomatic, meningovascular and meningeal neurosyphilis and of early tabes dorsalis. Poorer results naturally were obtained in cases of the tabetic form of dementia paralytica and of dementia paralytica. In 178 of the 200 cases treatment was started with a combination of routine and intraspinal therapy, and if there was no response then other measures were employed, particularly malaria therapy and treatment with tryparsamide. As must be expected, results in the treatment of syphilis of the central nervous system are measured in terms not of months but of years. Thus in only 4 patients with tabes dorsalis was there reversal of the condition of the spinal fluid to normal in less than a year. The longest period was eight years, and for the majority the period was one to three years. In cases of dementia paralytica the reactions were even more stubborn, and the longest period to reversal was thirteen years. Naturally it was not possible to achieve even this in all cases. Unfortunately, the authors have not found it possible to compare with other forms of therapy the treatment of a group of patients with syphilis of the central nervous system given routine and intraspinal therapy alone. Only such a report will really give an opportunity properly to evaluate its worth.

DR. HARRY M. ROBINSON, Baltimore: I am convinced of the usefulness of this method of treatment of syphilis of the central nervous system. During the early period of the use of intraspinal therapy I used this form, with serum, for almost all phases of syphilis of the central nervous system. As the

years rolled on, I gradually eliminated its use for all forms except tabes dorsalis and optic atrophy, as I had no good results with this treatment in cases of dementia paralytica. In cases of syphilis of the meningeal type my best results have been with the ordinary routine courses of injections of an arsenical of the arsphenamine group and a bismuth compound. I have had far better results with that than I have with intraspinal therapy. As a rule I prefer, when physical conditions permit, fever therapy in cases of uncomplicated syphilis of the central nervous system. I get much better results with that than with any other form of therapy. I believe, however, that it is preferable to start treating a patient with asymptomatic neurosyphilis with arsphenamine and a bismuth compound rather than to subject him to intraspinal therapy or fever therapy. If after a year or so there is not sufficient response one may resort to more strenuous forms of therapy. I would like to ask the authors a question which Dr. Cole mentioned. Has there been a control series of patients with syphilis of the meningeal type given just routine therapy, as it is commonly known?

DR. S. W. BECKER, Chicago: During the years that these patients were being treated I had the pleasure of administering the treatment to a great many of them. I can vouch for the efficacy of the form of therapy. When I went to the University of Chicago it was not possible to install the elaborate equipment necessary to administer intraspinal treatment safely, so that we had to try something else. We devised a method whereby after the initial treatment with sodium iodide given intravenously and soluble bismuth given six days a week for a few weeks we treated the patients, depending on whether they had early or late neurosyphilis, according to the base line schedule for the type of syphilis. After they had completed the arsphenamine phase of the therapy, instead of tapering the treatment off slowly, as is our custom, we gave these patients full doses of a bismuth compound in courses of ten weeks, only two months apart. We continued that therapy, sometimes for several years, until the reaction of the spinal fluid became negative. In an analysis of the results of this type of treatment prepared by Dr. Walsh and me and published in THE JOURNAL, Feb. 8, 1941, we found that our results were practically the same as those obtained by the Cooperative Clinical Group in the treatment of neurosyphilis. However, for our patients we had used collateral measures, such as fever therapy and the administration of tryparsamide in a small proportion, perhaps 15 per cent, as compared with something like 65 per cent for the patients of the Cooperative Clinical Group. We feel that this type of therapy is safe, convenient for the patient and apparently as efficacious as some of the more complicated types, but it must be carried out over a long period.

DR. A. BENSON CANNON, New York: I have been using this method of treatment of neurosyphilis since 1914. The more experience I have with it the more convinced I am of its value; in carefully selected cases no other treatment can supplant it. Sometimes in a case of neurosyphilis of six months' or a year's duration in which the reaction of spinal fluid has become positive only one or at the most two or three injections of serum are required to bring the reaction back to negative; thus the amount and number of doses of arsphenamine required in the subsequent period of treatment will be appreciably reduced. As to what Dr. Cole has said concerning the complicated technic of the Swift-Ellis method, I believe that the more experience one has with this treatment the more one can simplify it and that it can in reality be made an uncomplicated routine technic. One simplification which I have used for years is to allow the patient to go home at any time from fifteen minutes to an hour after he has had the puncture. My associates and I find that by thus making the routine puncture a simple procedure of examination we are able to obtain consent to puncture many more times than we could before we adopted this practice. This is particularly true in the clinic, where we now get consent to a spinal puncture when desired in practically 100 per cent of cases, compared to about 35 per cent previously. Another simplification I have adopted is in the manner of giving the intraspinal treatment itself. Whether it is given in the office or in the clinic I allow the patient to go home at any time from half an hour to two hours after the injection. I have never observed any serious accidents resulting from this practice. If the patients are carefully selected for this type of treatment it is safe to give the intraspinal injection in this fashion, thus doing away with the inconvenience to the patient of overnight hospitalization. One is accustomed to think of the spinal fluid as being more resistant to this type of treatment than the blood. As a matter of fact the reverse is frequently the case: giving intraspinal therapy to a patient whose blood and spinal fluid both show a strongly positive reaction will often render the reaction of the spinal fluid negative long before it has any effect at all on the blood.

DR. ROBERT R. KIERLAND, Rochester, Minn.: It must be remembered that no ideal system of treating syphilis of the central nervous system exists. This brings up the point brought out by Drs. Cole and Robinson regarding a control series. Unfortunately, few studies of any type of syphilis have adequate controls. Perhaps the only study which may be considered as such is that by Brusgaard. The ideal system would be to take 100 consecutive patients with neurosyphilis and treat them with nothing but the routine type of therapy in spite of what good or bad results may have been obtained. An additional hundred neurosyphilitic patients should receive nothing but a combination of intraspinal treatment plus routine therapy, the next hundred should receive tryparsamide, the next hundred fever therapy and so on. This is manifestly impossible, as when one treats private patients one tries to obtain the best results possible with a combination of all types of therapeutic measures. It must be remembered that in the majority of instances patients who were started on a routine therapy received no other type of therapy if they obtained good results. The same practice applied to intraspinal treatment. If while receiving a combination of routine and intraspinal treatment they obtained good results, other supplemental procedures were not used. The reason that many patients received these supplemental forms of treatment was that they did not do well with the type of therapy previously given. I think that will serve to clear up the remarks of Dr. Robinson. The results pictured on the slides are only those obtained before the institution of other therapeutic methods, and the complete paper will include also the final results obtained with all types of therapy. I am familiar with Dr. Becker's and Dr. Walsh's work concerning the prolonged use of bismuth compounds but have had no experience with it. If these studies are confirmed, it is a large advance in the treatment of syphilis of the central nervous system. I hardly agree with Dr. Robinson's remark that routine treatment alone does better than a combination of intraspinal and routine therapy. Certainly that is far from the experience of the Cooperative Clinical Group. This is borne out in practically all their studies, particularly the last three dealing with asymptomatic neurosyphilis, tabes dorsalis and the complete paper on symptomatic neurosyphilis.

The Plight of the Best.—Five years after graduation the ablest of my contemporaries were making $10,000 a year in practice and have continued at that level or above it. The equally able men who went into teaching or research were at $3,000 after six years and a few have worked up to $9,500 twenty years later. It may be replied that low salaries eliminate the incompetent. They do not. I may as well say straight out that the incompetents stick on. Low salaries may cause the money minded and the hard pressed to go elsewhere. The worst and the best stay on. One cannot pass over the incompetents as negligible. They clog the roads. But the plight of the best is truly distressing. Indeed, I could offer no excuses for what I know, but cannot reveal, regarding the struggles of some of the most eminent medical scientists in this rich country to raise and educate their none too numerous families. If you detect indignation in these remarks please remember that the best investigators in medicine are men of humility and modesty who do not know how to protect themselves in serving our highly competitive society. That seems to me society's loss, and its shame.—Gregg, Alan: The Furtherance of Medical Research, New Haven, Conn., Yale University Press, 1941.

ACUTE HEMATOGENOUS STAPHYLOCOCCIC OSTEOMYELITIS

TREATMENT WITH SULFATHIAZOLE WITHOUT OPERATION

WALTER A. HOYT, M.D.
ADRIAN E. DAVIS, M.D.
AND
GEORGE VAN BUREN, M.D.
AKRON, OHIO

Sulfathiazole, with its bacteriostatic and bactericidal properties, offers a new approach to the treatment of acute staphylococcic osteomyelitis by attacking the staphylococcus organism both in the blood stream and in the local lesion.

Up to the advent of sulfathiazole the treatment of the bacteremia or the toxemia has been directed toward supportive methods, such as infusions, blood transfusions and the use of antitoxins, as advocated by Baker and Shands [1] and other investigators. The universally accepted approach to the local lesion has been operative, varying from simple incision and drainage of the soft tissue abscess to more radical drilling or guttering of the bone. Authors are divided as to the extent and time of operation. One school, represented by such men as Hart,[2] Fraser,[3] Robertson [4] and Conwell and Sherrill,[5] advocates early incision of both bone and soft tissue. Another school advocates delayed operation and then only incision and drainage of the soft tissue (Wilensky,[6] Crossan,[7] Green and Shannon [8] and others). Both schools, however, have expressed the belief that some type of drainage is necessary. Attempts to combat the staphylococcus organism locally once drainage has been established have met with little success.

A review of studies of the end result of both acute and chronic osteomyelitis, regardless of the methods of treatment employed, is conclusive evidence of the inadequacy of the usual treatment of the disease. The high mortality, long hospitalization, severe deformities, secondary joint involvement, multiple operative procedures, economic loss and invalidism are grim monuments to this fact. Crossan [9] in 1938 made an excellent survey of all the literature on osteomyelitis for a seven and one-half year period and concluded that one fact was established clearly and one fact only; namely, the disease has a poor prognosis. Slightly more than one-fifth of the patients died and nearly two fifths of the survivors were crippled. Some of those not crippled were invalids or suffered periods of recurrent invalidism because of the disease.

In the recent literature there has been a definite trend toward conservative treatment, and the following significant observations have justified this conservatism. Early operation has increased toxicity, and negative blood cultures have become positive (Dolman [10]). The mortality appears to be greater among patients on whom operation is done early, especially when bone is opened (Pyrah and Pain [11]). Early operation does not appear to lessen the extent of involvement of the bone and may tend to increase destruction by disturbance of the blood supply.

Wilensky [6] has shown that foci of hematogenous osteomyelitis may subside completely and spontaneously without going on to the stage of necrosis and sequestration; that similarly sequestrated areas of bone can and do become revascularized and reincorporated with neighboring bone tissue in much the same manner as a bone graft. He likewise has expressed the belief that acute hematogenous osteomyelitis is an incident in a general bacterial infection:

> In the treatment of this disease in the early stages the most important item is the general infection. The ultimate outcome, death or recovery, depends entirely on this factor, and the mortality statistics of acute hematogenous osteomyelitis in its early stages reflect accurately the mortality of general bacterial infection. When divorced from the general bacterial infection and in the absence of any fatal complications or associated lesion, the mortality of the local osseous lesion is nil.

Since the advent of sulfathiazole the literature has been voluminous in reports of both the experimental and the clinical uses of the drug. Barlow and Homburger [12] have shown that sulfathiazole has definite bacteriostatic and bactericidal effects on the staphylococcus, both in vivo and in vitro. They have demonstrated also that the life of experimental mice infected with highly virulent strains of Staphylococcus aureus is prolonged and that abscesses in the kidneys and other organs are prevented or healed by the use of sulfathiazole.

Repeated reports have established the fact that sulfathiazole definitely sterilizes the blood in staphylococcic septicemia, effecting a cure.[13] Local lesions caused by the staphylococcus have also shown favorable results from internal administration of the drug.

Sulfathiazole is being used generally in the treatment of both acute and chronic osteomyelitis, but to date few reports have appeared. Dickson and his associates [14] have used it both internally and locally in a series of cases of chronic osteomyelitis, with favorable results. Melton [15] reported 10 cases of acute osteomyelitis in which it was used internally but in association with incision and drainage. He stated that it did not lessen the need for surgical drainage or check the progress of suppuration. It did lessen toxemia.

To date there have been no published reports of internal treatment with sulfathiazole without drainage of the local abscess. Nachlas [16] has treated a series of patients

From the Children's Hospital.
Read before the Section on Orthopedic Surgery at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 5, 1941.

1. Baker, L. D., and Shands, A. R.: Acute Osteomyelitis with Staphylococcemia, J. A. M. A. **113**: 2119 (Dec. 9) 1939.
2. Hart, V. L.: Acute Hematogenous Osteomyelitis in Children, J. A. M. A. **108**: 524 (Feb. 13) 1937.
3. Fraser, J.: Acute Osteomyelitis, Brit. M. J. **2**: 539, 1934.
4. Robertson, R. C.: Acute Hematogenous Osteomyelitis: Analysis of Seventy-Five Cases, J. A. M. A. **107**: 1193 (Oct. 10) 1936.
5. Conwell, H. E., and Sherrill, J. D.: Acute Osteomyelitis in Childhood: Diagnosis and Treatment, South. M. J. **30**: 171, 1937.
6. Wilensky, A. O.: Acute Hematogenous Osteomyelitis: Classification of Cases of Acute Hematogenous Osteomyelitis as Determined by Therapeutic Indications, Arch. Surg. **34**: 320 (Feb.) 1937.
7. Crossan, E. T.: Conservative Treatment of Hematogenous Osteomyelitis, Ann. Surg. **103**: 605, 1936.
8. Green, W. T., and Shannon, G.: Osteomyelitis of Infants: A Disease Different from Osteomyelitis of Older Children, Arch. Surg. **32**: 462 (March) 1936.
9. Crossan, E. T.: Hematogenous Osteomyelitis: Collective Review of the Literature from 1932 to 1937, Surg., Gynec. & Obst. **68**: 176, 1938.
10. Dolman, E. E.: Pathogenic and Antigenic Properties of Staphylococcic Toxin, Am. Pub. Health J. **23**: 125, 1932.
11. Pyrah, L. N., and Pain, A. B.: Acute Infective Osteomyelitis: Review of Two Hundred and Sixty-Two Cases, Brit. J. Surg. **20**: 590, 1933.
12. Barlow, O. W., and Homburger, E.: Specific Chemotherapy of Experimental Staphylococcal Infections with Thiazol Derivatives of Sulfanilamide, Proc. Soc. Exper. Biol. & Med. **42**: 795, 1939.
13. Homburger, M., and Ruegsegger, J. M.: The Treatment of Staphylococcal Septicemia with Sulfamethylthiazole and Sulfathiazole: Report of Twelve Cases, Ann. Int. Med. **14**: 1137, 1941.
14. Dickson, F. D., and others: The Treatment of Chronic Osteomyelitis by the Local Use of Sulfathiazole, read before the American Academy of Orthopedic Surgeons, New Orleans, 1941, to be published.
15. Melton, George: Sulfathiazole in the Treatment of Staphylococcal Infections, Lancet **1**: 274, 1941.
16. Nachlas, I. W.: Personal communication to the authors.

in this manner without any operative procedure and reported excellent results. His work will appear in the literature at an early date. Badgley [17] has treated several patients with sulfathiazole, delayed incision and drainage with good results. He has also treated a secondary bone lesion in 1 of his cases without surgical intervention.

Since August 1940, in 8 consecutive cases of acute osteomyelitis treatment with sulfathiazole without operation has been given at the Children's Hospital. The unusual course of the disease and the abscess, contrary to all previous experience and associated with end results superior to anything that we have heretofore seen, leads us to review the 8 cases.

METHOD OF TREATMENT

The treatment here outlined should be undertaken only in a hospital, under the most competent supervision. On admission supportive treatment is given, and the pain is controlled by immobilization and the administration of mild sedatives. Oral sulfathiazole therapy is instituted immediately. The initial dose is determined by the body weight of the patient. A safe dose for initial administration is 1 to 1½ grains (0.06 to 0.1 Gm.) per pound (0.5 Kg.) per day. For a child of 100 pounds (45.4 Kg.) the initial dose would be 20 grains (1.2 Gm.) every four hours. Subsequent dosage is regulated by determination of the blood level of the drug. An attempt is made to maintain the blood level at 4 to 6 mg. per hundred cubic centimeters throughout the treatment. To accomplish this, one must give the drug every four hours because of rapid excretion. An equal dose of sodium bicarbonate is given simultaneously.

Since on admission patients with acute osteomyelitis are dehydrated, liquids are immediately forced and saline solution is given intravenously. The procedure is repeated as often as necessary to maintain a normal relation of intake and output of fluids. While the forcing of liquids may lower the blood level of the drug, it is better to increase the dose than to decrease the intake of fluids. Renal complications if present are the result of crystalline deposits in the tubules of the kidney, and concentration and acidity of the urine are to be avoided. Administration of sulfathiazole is continued unless contraindicated for at least two to five weeks at full dosage. The duration of therapy is determined by the condition of the patient and the course of the disease.

17. Badgley, C. E.: Personal communication to the authors.

It is advisable to give transfusions of 300 to 500 cc. of whole blood every few days during the first two weeks and at less frequent intervals thereafter, depending on the condition of the patient.

The treatment of the local lesion is that of nonintervention. Wet packs have been employed, but they are of questionable value. Immobilization during the painful stage of the abscess is advised and is best carried out by posterior splinting or extension. The duration must be determined by the condition in the individual case. Obviously, for extremities with extensive involvement of bone and danger of spontaneous fracture, immobilization must be employed over a longer period of time. As a rule, however, after the abscess begins to recede, rigid immobilization should be discontinued and passive and active motion of the part should be started. This results in less atrophy and better repair. The local lesion is protected against trauma by thick dressings, as spontaneous rupture of the abscess is to be avoided.

Care after discharge from the hospital is likewise dependent on the condition in the individual case. The same methods of protection as are employed in any other type of treatment should be used, but the problem is simplified because there is no draining sinus. Weight bearing with protection is instituted as early as the local lesion will safely permit it.

TABLE 1.—*Summary of Cases*

Patient	Age	Sex	Onset	Cutaneous Lesion	Organism	Lesion	Blood Cultures	Abscess Cultures	Hospital Days	End Result
1. B. M.	11	♂	August 1940	Infected bite	Staph. albus	Scapula, humerus	+++	+	66	Limitation of motion of shoulder; no drainage
2. R. J.	11	♂	July 1940	None	Staph. aureus	Femur, tibia	+++	—	54	Normal function; no drainage
3. L. A.	6	♀	September 1940	Furunculosis	Not determined	Tibia	—	—	21	Normal function; no drainage
4. R. B.	14½	♂	September 1940	Furunculosis	Staph. aureus	Clavicle, femur	+	++	39	Normal function; no drainage
5. J. A.	7	♂	November 1940	None	Staph. aureus	Fibula	—	++	39	Normal function; no drainage
6. N. G.	14	♀	December 1940	Infected blister	Staph. aureus	Right ilium	++	—	65	Slight tilting of pelvis; no drainage
7. R. A.	13	♂	December 1940	Furunculosis	Staph. aureus	Left ulna	++	+	120	Limitation of motion of elbow; draining sinus of arm
8. J. B.	10	♂	February 1941	Furunculosis	Staph. aureus	Metatarsal, great toe	—	+	33	Normal function; no drainage

REVIEW OF CASES

Table 1 gives a summary of the data in the 8 cases reported. In 6 there was a history of some previous cutaneous infection, the most common lesion being furunculosis (4 cases). The staphylococcus organism

Fig. 1 (case 7).—Osteomyelitis of shaft of ulna.

was demonstrated in 7 cases either by blood culture or by aspiration of the local abscess. In case 3 no organism was demonstrated.

Repeated blood cultures were studied in all the cases. In 5 of these, cultures of material obtained at some time during the first week and a half were positive. After this period no positive reports were returned, although

blood for culture was taken every few days during the hospital stay. The colony count varied from ninety per field (case 1) to three to four (case 8).

The local abscess was aspirated early for diagnosis in 5 cases, and culture of the aspirated material was reported positive. In 3 of these cases aspiration was

Fig. 2 (case 1).—Appearance ten months after onset of osteomyelitis.

repeated three to five weeks later, and the cultures were negative. The late aspirations were made after rather long administration of sulfathiazole.

Leukocytosis, the white cell count varying from 40,000 to 15,000, was present in all the cases during the early stage of the disease. In several cases there was a fall in the red blood cell count and the hemoglobin content during the second week. The urine remained normal in the whole series throughout the hospital stay. Frequent determinations of the blood sulfathiazole level were made. The highest level in any case was 14 mg. per hundred cubic centimeters (case 6). In case 3 2.8 mg. was the highest level that could be maintained. The average for the series was 4 to 6 mg. per hundred cubic centimeters during active administration of sulfathiazole.

For the first two weeks the local soft tissue lesions presented the same appearance as any other acute inflammatory abscess. Then they became less hot and tense and smaller and were less painful. In three to four weeks the inflammatory area if still present became darker and did not feel hot, and an indurated border and a soft, rubbery center developed. As time progressed, the local lesion completely disappeared and the skin took on its normal appearance.

In case 7, in which a most extensive involvement of the ulna and the forearm occurred, the local lesion remained closed for four months. There persisted over the forearm three discolored areas with soft centers. After being struck with a toy these lesions started to drain seropurulent fluid from which Staph. aureus could be cultured. This drainage persists, although it appears superficial and does not seem to lead to bone.

The symptoms in this series at onset were similar to the symptoms in any other group of cases of acute staphylococcic osteomyelitis. There were severe toxemia, a high temperature and swelling and pain in the extremities. In case 1 the most severe systemic reaction in the whole series occurred. There was a positive blood culture of ninety colonies per field, and the osteomyelitis was complicated with a chest condition producing material from which Staphylococcus albus could be cultured. The patient was treated in an oxygen tent for ten days and had the severest soft tissue involvement of the shoulder, neck and face that we have ever encountered. Any type of operative treatment was contraindicated. Considered moribund, the patient was treated symptomatically and given sulfathiazole. In the third week of treatment two small exploratory incisions were made in the skin over the scapula. No pus was encountered, and the incisions were closed with sutures. Primary healing took place, and cultures of material from this area were sterile. The favorable end result of treatment in spite of the general condition, the roentgen appearance and the local soft tissue lesions led us to treat subsequent patients with sulfathiazole without operation. Many of the other patients presented severe general and local symptoms and responded equally well.

The majority of patients showed little improvement until the second week. The temperature remained ele-

Fig. 3 (case 6).—Osteomyelitis of right side of ilium: appearance five months after onset.

vated for two to six weeks and in several cases was higher in the second and third weeks than in the first. Even though the temperature remained elevated this late and the local lesion was still present, the general condition and the toxemia were much improved. The patients were bright and alert and required no medication for the local lesions.

Roentgen studies made at onset in all cases followed by frequent rechecks up to the present have shown extensive bone changes. The primary bone lesion has progressed at first much as it does in similar cases in which treatment is by incision and drainage without sulfathiazole. Areas of absorption have been followed by bone proliferation and in 3 cases by definite sequestration (cases 5, 7 and 8). As time has passed the areas of sequestration in cases 5 and 7 have disappeared and have become incorporated into the substance of the ulna and of the fibula respectively. In case 7 the whole shaft of the ulna was involved (fig. 1). Two months later an unusual amount of proliferation appeared, increasing the diameter of the bone. This at first cast a faint shadow but later became more calcified and gave the appearance of an involucrum. At the end of six months a decrease in the size of the bone occurred, with reestablishment of a more normal contour. In case 1 enormous bone proliferation involved the whole bone (fig. 2), and this has persisted, although it has taken on more nearly the appearance of normal bone. In case 4 there was little bone proliferation in the end clavicle, and studies of the end results reveal a well healed lesion. In case 6 the disease of the ilium has progressed like any other osteomyelitis (figs. 3 and 4) except that the involvement is not as extensive as that usually found. In case 3 the disease was confined to the upper end of the tibia and after eight months has almost completely disappeared.

Fig. 4 (case 6).—End result.

In cases in which more than one bone was involved little bone proliferation is observed in the secondary lesion, and after six to eight months the lesions are well healed. In case 2 there was a secondary lesion of the epiphysis of the femur and of the patella, but healing appears to have taken place.

End results are considered from the standpoint of the general condition and the local lesions. The excellent physical condition of the patients is impressive. Asymptomatic after two to three months, they have the appearance of normal, healthy children, have gained weight and have normal blood counts. Short hospitalization and immobilization, with early return to normal activity without plaster casts or draining sinuses, have produced none of the usual appearances of the septic, anxious patient who has been operated on for osteomyelitis.

The end results of treatment of the local lesion have been most satisfactory to date. In only 2 cases has any drainage developed. In case 1, eight months after onset a serous drainage developed in the site of the incision in the skin over the scapula. This was superficial and persisted for only two weeks. Culture of material obtained from this area was not positive. In case 7 the lesion is still draining after a direct trauma to the region of the ulna, and culture shows Staph. aureus. In 6 of the 8 cases there has been an increase in size of the part affected. This is evident both to inspection and by measurement but is becoming less apparent as time goes on. Only 2 patients have shown any limitation in function. Patient 1 (fig. 2) has limitation in rotation of the shoulder of about 50 per cent. Abduction of the shoulder is limited to 45 degrees. There is also definite muscular weakness of the shoulder girdle. Patient 7 at this time has almost complete limitation in pronation and supination of the forearm, and flexion and extension of the elbow and the wrist are diminished about 50 per cent. Patient 6 has slight tilting of the pelvis. The progress of the osteomyelitis, data on sulfathiazole therapy and the end result in case 5 are shown in figures 5, 6, 7 and 8.

Practically no atrophy has resulted in the region of the local lesion, and the color and the character of the skin have returned to normal. There appears to be no evidence of induration except in patient 7, whose lesion is draining.

Of the 8 patients treated, only 1 remains in the hospital at this time. The longest hospital stay has been one hundred and twenty days and the shortest twenty-one days, with an average of fifty-four days for the series. With increasing experience in the treatment of the disease we believe that the hospital stay can be considerably reduced. Seven patients have returned to school. Two are using crutches for protection, 1 of whom has a light foot cast to prevent weight bearing.

COMPLICATIONS

Children tolerate sulfathiazole well. Fortunately acute osteomyelitis occurs mostly in children and young adults. To date we have had no complication which has made it impossible to carry out the required treatment. In 2 cases a cutaneous eruption appeared simulating erythema nodosum. In 1 of these the drug was withdrawn for two days, the condition cleared up and use of the drug was resumed. In the other the drug was not withdrawn and in three days the cutaneous

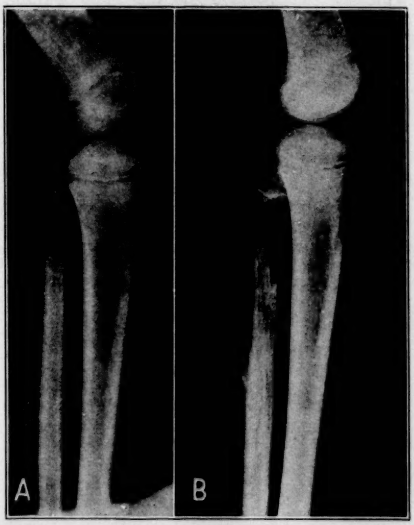


Fig. 5 (case 5).—Progress of osteomyelitis: *A*, seven days, *B*, twenty-three days after onset.

condition disappeared. There were no renal complications. Accurate studies of the intake and output of fluid were made, and the amounts remained well within normal range in all cases. In case 7 after prolonged use of the drug over several weeks conjunctivitis developed, necessitating discontinuance. In several cases some nausea was encountered during the first few days but never was a serious problem. In 2 cases we found that the temperature remained elevated until administration of the drug was discontinued. This may be true oftener than is realized. We do not hesitate to discontinue administration of the drug at any time when in doubt as to a complication. Few serious complications of the drug's use have been reported. Long

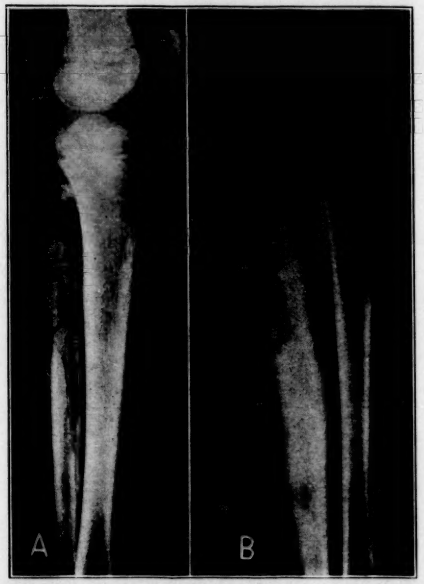


Fig. 6 (case 5).—Further progress: *A*, six weeks, *B*, six months after onset.

and his associates [18] have described in detail the complications produced by sulfathiazole and its toxicity as compared with that of sulfapyridine and sulfanilamide.

COMMENT

Osteomyelitis is primarily a bacteremia, and the bone lesion is secondary. Nussbaum [19] showed that the terminal arteries at the metaphysis are dilated, forming a so-called venous lake. Here stasis can occur and bacterial masses as large as 20 microns can lodge. With the slowing of the blood stream, lessened phagocytosis occurs and favorable conditions for an inflammatory process arise. This extends to the periosteum and later, after a certain amount of elevation, breaks through into the soft tissue. If undisturbed surgically the abscess ruptures spontaneously, creating a sinus directly to the bone. When incision and drainage are done, either early or late, the same situation is artificially produced. When operation is used in a case of acute osteomyelitis it follows that there must be some disturbance in the blood supply to the bone and the soft

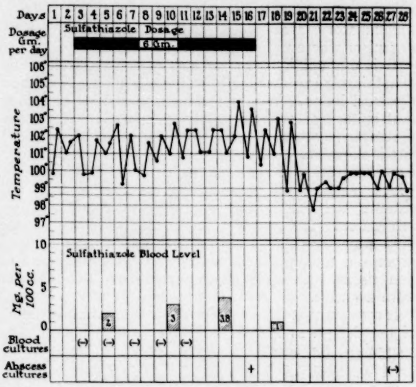


Fig. 7 (case 5).—Clinical course in hospital.

tissues as a result of the operation. Also, when continued drainage or packing is employed in the after-treatment there is sure to be a mixed infection involving the whole inflammatory area. It is difficult to evaluate

Fig. 8 (case 5).—End result.

how much these two factors have affected the acute and chronic course of the disease in the past.

It has been shown that when sulfathiazole is administered there is a definite blood concentration which can be maintained at a desired level. It has also been shown that material aspirated from an abscess of acute

18. Long, P. H.; Haviland, J. W.; Edwards, L. B., and Bliss, E. A.: The Toxic Manifestations of Sulfanilamide and Its Derivatives, J. A. M. A. **115**: 364 (Aug. 3) 1940.
19. Nussbaum, A.: Beziehungen der Knochengefässe zur akuten Osteomyelitis, Zentralbl. f. Chir. **49**: 700, 1922.

osteomyelitis treated by sulfathiazole will show a concentration as high as if not higher than that of the blood taken at the same time. We think it can be assumed from these observations that there is a diffusion of sulfathiazole into the whole inflammatory area as well as into the blood. The maintenance of a sulfathiazole concentration in both the blood and the inflammatory area would attack the staphylococcus organism wherever it might be.

It is our opinion that in cases of acute osteomyelitis sulfathiazole can eventually sterilize both the blood and the local lesion. We have shown on several occasions that culture of material aspirated early is positive for the staphylococcus and that culture of material aspirated late is negative. As further evidence, there came under our observation a case not included in this series in which incision and drainage of an abscess over the scapula was made after four weeks of sulfathiazole medication. No pus was found, but a yellow serous fluid was present. There was evidence of a healing process, a copious blood supply and excessive granulation tissue. Cultures of material from this abscess were negative.

It appears that in our series the earlier administration of the drug was started after the first symptom was recognized the shorter was the period of elevation of the temperature and the less the involvement of bone not be drawn, but there are evident improvements over previous end results. Only ten months have elapsed since the onset of acute osteomyelitis in the first patient treated, and what the ultimate result will be after one, two or ten years cannot be predicted. Comparison of these patients with any other series of patients, however, shows striking differences. There has been no mortality; hospitalization and disability have been shorter; there have been no draining sinuses except late in 1 case; no operations have been performed, either primarily or secondarily; deformities and limitation in motion have been minimal. The patients are not invalids and with the exception of 1 still under treatment are carrying on normal activity and attending school.

The most revolutionary part of the treatment suggested in this paper is the handling of the local lesion without operation. Surgeons trained under the precept and surgical principle "Where there is pus, drain" find it extremely difficult to refrain from surgical intervention. However, by waiting we have repeatedly seen painful, red, hot, fluctuating abscesses, from which pus which produced positive cultures could be aspirated, change into painless, pale, soft, doughy areas which yield negative cultures and finally completely disappear.

The second week is the most difficult time to pass without resorting to surgical procedures. The use of sulfathiazole produces no immediate, spectacular results. The bacteremia and toxemia of the patient will improve rapidly, but the elevation of temperature will continue, and there may be at first an apparent increase in the abscess. Continued elevation of the temperature and an increase in the abscess during the second week are not an indication for surgical procedures, however.

TABLE 2.—*Data on Sulfathiazole Therapy*

Patient	Age	Sex	Lesion	Onset of Symptoms	Beginning of Therapy	Daily Dose	Days of Treatment	Blood Level, Mg. per 100 Cc.	Appearance of Normal Temperature
1	11	♂	Scapula, humerus	8/17/40	5th day	6 Gm.	45	2.5 to 7	42d day
2	11	♂	Femur, tibia	7/10/40	3d day	6 Gm.	14	5.5 to 5.8	14th day
3	6	♀	Tibia	9/17/40	13th day	6 Gm.	41	4.4 to 5.7	14th day
4	14½	♂	Clavicle, femur	9/28/40	3d day	4 to 10 Gm.	37	3.3 to 8.3	30th day
5	7	♂	Fibula	11/ 5/40	8th day	6 Gm.	14	1.0 to 3.8	20th day
6	14	♀	Ilium	12/20/40	24th day	6 Gm.	45	3.5 to 14	56th day
7	13	♂	Ulna	11/ 4/40	10th day	3 Gm.	14	1.7 to 4	21st day
8	10	♂	Metatarsal	2/15/41	10th day	3 Gm.	34	2.3 to 9	13th day

(table 2). We have observed in the last ten months 3 patients with positive blood cultures who had tenderness and pain over the long bones. They were given sulfathiazole within two or three days after the onset of symptoms. No local abscess occurred, and there was no roentgen evidence of involvement of bone. Obviously these were not included in our series, but we feel it is possible that these 3 patients had acute osteomyelitis which because of the early administration of the drug did not follow the usual course.

From the study of the roentgenograms in this series it does not appear that the extent of the involvement of bone has been lessened. On the contrary, the typical degenerative changes of acute osteomyelitis have been followed by sequestration and the formation of an involucrum, with ultimate excessive bone proliferation. As time has elapsed this proliferation has decreased, much as does a callus about a fracture. Regardless of how soon the temperature returned to normal or how soon the drug was given after the onset of the disease, these bone changes have occurred. We agree with Long,[20] Wilensky and other investigators that the sequestrums if sterile become revascularized and act as a bone graft.

The disadvantages of previous methods of treatment for acute osteomyelitis have been a high mortality, long hospitalization, persistent drainage, multiple secondary operations, deformities and invalidism.

Our series of patients treated by sulfathiazole without operation is not large, and too many conclusions should

We object to incision and drainage with the use of sulfathiazole because the blood supply to the bone and the surrounding tissue is disturbed, interfering with the diffusion of the drug. With a draining sinus it is impossible to maintain a high concentration of sulfathiazole. Operation inevitably is followed by secondary infection through the draining sinus, producing the same set of conditions so objectionable in the older methods of treatment. The goal of this treatment of staphylococcic osteomyelitis by sulfathiazole without operation is the sterilization of both the blood and the local lesion. If that can be accomplished the bacteremia and toxemia will be controlled and repair in the bone and soft tissue will proceed normally.

CONCLUSIONS

1. In 8 cases of acute staphylococcic osteomyelitis in which treatment with sulfathiazole without operation has been given the end results after 10 months appear to be better than results obtainable by other methods.

2. The bacteremia, as evidenced by positive blood cultures, was controlled by the administration of sulfathiazole without incision and drainage of the local abscess.

20. Long, P. H.: Personal communication to the authors.

3. Abscesses have occurred of which cultures at first were positive for staphylococci but after sulfathiazole administration were negative.

4. Acute fluctuating staphylococcic abscesses have completely disappeared without incision and drainage.

5. Sulfathiazole does not appear to decrease the involvement of bone in primary lesions. In some cases there has been an increased proliferation.

6. Secondary bone lesions show fewer changes and return more quickly to normal appearance.

7. Sequestration may occur and when it does appears to act as a sterile bone graft.

8. There were no deaths in the series.

9. Treatment by this method must be instituted in a much larger series of cases and a longer period must elapse before final evaluation of its merits can be ascertained.

AUTHORS' NOTE.—Since the presentation of these eight cases in June, additional patients have been treated without operation with similar results. These include two cases of septic arthritis of the hip with bone changes. There have been no recurrences in any of the cases, and seventeen months has elapsed since the first case was treated.

407 Ohio Building.

ABSTRACT OF DISCUSSION

DR. CLARENCE H. HEYMAN, Cleveland: I have had no experience with this method of treatment as described by Drs. Hoyt and his collaborators. This is further evidence of the trend toward conservative treatment of acute osteomyelitis. It is difficult to keep hands off when we have been brought up on the axiom that where there is pus surgical drainage is required. Even with simple bone drilling it has been my experience that the progress of the disease and bone destruction with sequestration continue. Dr. Greene of the Children's Hospital of Boston not long ago gave us courage to delay any sort of operative treatment in very young children until the formation of a localized abscess. However, we recognize that in infants the infecting organism as a rule is the streptococcus rather than the staphylococcus. The authors now go still further in keeping hands off, but with the addition of sulfathiazole. They are to be congratulated on their courage. After studying the material in their exhibit I have been sufficiently impressed to adopt this method at least for a trial.

DR. CARL E. BADGLEY, Ann Arbor, Mich.: In February I showed Dr. Hoyt a case of osteomyelitis of the ilium associated with a pyarthrosis of the hip, which was treated by our usual procedure with incision and drainage of the involved area, with drainage of the abscess of the iliac fossa and drainage of the pyarthrosis, followed by sulfathiazole, with the wound kept open by petrolatum gauze. There was a prompt dropping of temperature. The child convalesced rapidly, so that an early secondary closure with sulfathiazole in the wound was followed by healing of the wound and with excellent range of motion in the hip joint. This child has an excellent hip. He has no drainage. The wound has healed and is doing very well. This has been our standard method of treatment for acute hematogenous osteomyelitis. The authors have presented something that should not be considered as a proved method of treatment but something that should be utilized from an experimental point of view. We should have an open mind and if possible see if this is not a good method of treatment. I feel convinced, as Dr. Key does, that the open operation plus the local implantation of sulfathiazole is a good procedure. Can we get away from that procedure? Can we, by this oral administration, have these patients get along without the unsightly scars? I don't think that Dr. Hoyt and his co-workers have proved that yet. They have proved that the 8 cases in which they operated did. The cases we have presented have done quite well, but that is not proof that patients can be treated for acute osteomyelitis by this method successfully in large numbers.

DR. LENOX D. BAKER, Durham, N. C.: In certain strains of staphylococcus, one or more of the sulfonamide drugs may be specific. Recently Miss Mary Poston of our bacteriology department ran in vitro determinations on the effectiveness of the sulfonamides on fifty-seven strains of staphylococcus. The studies were made on broth cultures with various concentrations of the drugs up to 10 mg. I am aware that in vitro studies will not tell us all we want to know, but they do help in determining the susceptibility of the organism. Of the fifty-seven strains, twenty-eight were not affected by any of the drugs; twenty-nine were affected by one or more of the drugs, but only eight of the twenty-nine were affected by what we took as a maintainable blood level of sulfathiazole for an average person, that is, 5 mg. per hundred cubic centimeters. Of the eight strains affected by sulfathiazole, four were affected also by one or more of the other sulfonamides. Seven of the 8 cases which Drs. Hoyt, Davis and Van Buren have presented came from the same locality and their infections occurred between August 1940 and February 1941. Of the 7, 6 had some kind of cutaneous lesion. Could these patients have had a common infection of staphylococcus which was susceptible to sulfathiazole? If so, naturally one would expect the results which have been shown. My associates and I have treated 15 cases of staphylococcic septicemia with sulfathiazole. In some of the cases the results have been most satisfactory but evidently not all strains of this organism are affected by the sulfonamides. As to incision and drainage interfering with the blood supply, the thing that makes the staphylococcus such a potent organism is its toxin, which has four major actions: it coagulates plasma, it necrotizes tissue, it destroys young leukocytes and it hemolyzes red cells. We all have had the same experience in operating on acute osteomyelitis. As we make our incision there are a few skin bleeders, but the remainder of the tissues have no real capillary bleeding, as the plasma is coagulated. Dr. Key states that when he makes sulfathiazole determinations on the pus it usually runs somewhere less than 50 per cent of what the concentration is in the blood, so like Dr. Key I feel that it is a good idea to drain the abscess and put the sulfathiazole in the wound.

DR. FREDERICK A. JOSTES, St. Louis: Dr. Alexis F. Hartman, professor of pediatrics at Washington University School of Medicine and chief at St. Louis Children's Hospital, has treated acute osteomyelitis with chemotherapy for a period of two years. It was my privilege to see most of these cases through the entire process. I have also treated some adults. It is my belief that the results obtained by judicious administration of the sulfonamide drugs locally and by mouth with early incision, when aspiration fails to define the organism causing the infection, are as good both roentgenologically and in the course of the illness as they are in those cases in which incision is not made and that these cases afford less hazard to the patient and worry to the doctor. We have a rather large proportion of cases in which Streptococcus hemolyticus and not the staphylococcus was the cause. Local application of the necessary drug was made after incision as well as other forms of administration to keep up the blood concentration sufficiently long not only to stop the spread of infection but to prevent recurrences, which are so frequently seen because the drug in small doses was not given over a sufficiently long period of time after the temperature and symptoms subsided. At no time were bones drilled or was the cortex broken surgically. Our x-ray results are not very different from those of Dr. Hoyt and his co-workers. Sclerosing was noted in most of the cases. In my own cases I incised early—early meaning when we got the cases within thirty-six or forty-eight hours—the drug was put into the area as well as given by mouth. We irrigated many of the old abscesses, and in many cases even though the temperature had been normal for some time the abscess was not sterile until after irrigation with the drug solution.

DR. WALTER A. HOYT, Akron, Ohio: We were impressed with the pathologic condition in 1 case which was seen simultaneously with the first case treated. This case, one of osteomyelitis of the scapula, had been treated by sulfathiazole without operation for four weeks before admission. The patient was afebrile and asymptomatic and examination showed a dough-like swelling over the scapula. Incision and drainage were done but pus was not encountered. There was present a serosanguineous fluid, and it had the appearance of granulation tissue with a copious blood supply. Culture from this area was negative. We have encountered many men this week in our exhibit who have been

working with sulfathiazole. We have had a good cross section of what is going on over the country in this work. It has been interesting to hear a description of the pathologic condition found when operation has been performed late in the disease. Dr. Nachlas of Baltimore has treated quite a series of cases in the manner we have suggested without operation. Dr. Perrin Long feels that we are justified in continuing with this type of treatment. It is going to take men like Dr. Baker and a great deal of investigative work to stabilize the method that is to be followed as far as osteomyelitis is concerned. Dr. Key said he knew a patient who had died after sulfathiazole treatment. I have seen many patients die after operation, and I believe that many of them have died because they were operated on too early. The most convincing part of the whole treatment is the appearance of the patients. They are healthy and have returned to school and normal activity. They have no draining sinus, no atrophy, no loss in range of motion, no pain and no "smell." We are going to continue conservatively. We want to see what these cases are going to do after one year, two years, three years and longer after onset. We do not expect 100 per cent results. We expect that in some of these cases drainage will occur. We are going to report on these cases later. If any physicians treat any cases of osteomyelitis in the manner described by us, we would appreciate hearing from them. Dr. Van Buren, our surgical resident at Children's Hospital, is responsible largely for starting this work, and Dr. Roberts and the other members of the staff made it possible for us to carry it on.

SPONTANEOUS PNEUMOMEDIASTINUM IN THE NEWBORN

BERNARD GUMBINER, M.D.
AND
MEYER M. CUTLER, M.D.
CHICAGO

It has been stated that more than one third of all deaths of infants occur during the first two weeks of life.[1] Asphyxia and atelectasis are listed second only to prematurity as the cause of death in the neonatal period.[1] Few symptoms are the source of more concern to the pediatrician than dyspnea and cyanosis in the newborn infant. In many instances, although definite abnormal physical signs in the heart and lungs are not elicited, a diagnosis of congenital atelectasis is made. In some cases the symptoms become progressive and lead to death despite therapeutic measures such as clearing of the air passages, administration of oxygen, stimulation and blood transfusions. Many physicians when confronted with dyspnea and cyanosis in the newborn have had a feeling of inadequacy from the aspect both of accurate diagnosis and of adequate treatment.

This sense of inadequacy can be relieved in part by recognizing spontaneous pneumomediastinum as a possible cause of these alarming symptoms, by diagnosing it when it is present and by instituting appropriate treatment. It is hoped that the description of the diagnostic and therapeutic procedures will serve to prevent some of the tragic deaths in the neonatal period.

Cases of pneumomediastinum or mediastinal emphysema in the newborn have been described before, but the reports have been few and their real import not recognized. In 1928 Stransky [2] reviewed the literature on pneumomediastinum and discovered that as early as 1853 Guillot [3] had reported 2 cases of the condition in the newborn, the diagnosis in both having been made at autopsy. We have been able to find reports of only 8 additional cases since that time: 2 cases reported by Schuler [4] and 1 each by Kirchgessner,[5] Faber,[6] Rosenblum,[7] Poeck,[8] DeCosta [9] and Fisher.[10]

No mention of the syndrome is made in general pediatric textbooks or treatises on the newborn in common use. Failure to recognize the condition would seem to be due to the fact that an adequate clinical description of the syndrome and a recognition of its importance have been lacking. Recent animal experimentation by C. C. Macklin [11] and by Fisher and Macklin [12] and a description of the syndrome in adults by Hamman [13] have served to bring the subject to the attention of the medical profession once again. None of the reports which we have found have stressed sufficiently the pathognomonic appearance of the lateral roentgenogram,[14] and as far as we could determine no one has ever employed simple therapeutic aspiration.[15] It is these measures, essential for diagnosis and therapy, that we wish to stress most in our presentation.

In March 1940 we observed our first case of mediastinal emphysema in the newborn infant, and it was the dramatic result obtained in this instance that prompted us to look for more cases and to present this report.

The patient was a boy weighing 9 pounds (4.1 Kg.), born by normal delivery at the Michael Reese Hospital. He required no resuscitation at birth, his cry was good and there was no dyspnea or cyanosis. About thirty hours after birth he suddenly became dyspneic and cyanotic. Examination disclosed the following essential features: Severe cyanosis was present, with distention of the veins of the neck. The respiratory rate was more than 130 per minute. The heart tones were distant, and no murmurs could be heard. No abnormal breath sounds could be made out, although there was some suppression of sounds over the posterior portion of the chest. There was no subcutaneous emphysema and no bulging of the precordium.

The infant was placed in a Hess oxygen bed, and stimulants were administered. An emergency roentgenogram showed, in the posterior-anterior view (fig. 1), distortion of the cardiac

From the Pediatric and Roentgenologic departments of Michael Reese Hospital.

Read before the Section on Pediatrics at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 4, 1941.

Dr. Gumbiner is associate attending physician of the Children's Division, Cook County Hospital, and formerly was resident physician of the Sarah Morris Hospital; Dr. Cutler is resident roentgenologist, Michael Reese Hospital.

The following physicians gave permission to include in this report cases from their private and ward services at Michael Reese Hospital: Drs. Philip Rosenblum, Arthur Abt, Carl Cohen and Emanuel Padnos. Dr. Henry G. Poncher permitted inclusion of the case from the tuberculosis service of the Cook County Hospital.

1. Holt, L. E., and McIntosh, Russel: Holt's Diseases of Infancy and Childhood, ed. 11, New York, D. Appleton-Century Company, Inc., p. 56.

2. Stransky, Eugene: Beiträge zur Klinik des mediastinalen Emphysems durch Alveolarruptur im Säuglingsalter, Monatschr. f. Kinderh. **39:** 104-112, 1928.

3. Guillot, N.: Observations d'emphysèmes, Arch. gén. de méd. **2:** 151, 1853.

4. Schuler, F.: Ueber partiellen Spontanpneumothorax des Neugeborenen, Arch. f. Kinderh. **113:** 160, 1938.

5. Kirchgessner: Ueber einen Fall von subkutanem Emphysem beim Neugeborenen, München. med. Wchnschr. **51:** 455, 1904.

6. Faber, H. K.: Subcutaneous Emphysema in an Infant Three Days Old, Am. J. Dis. Child. **19:** 388 (May) 1920.

7. Rosenblum, Phillip: Subcutaneous Emphysema, Am. J. Dis. Child. **32:** 159 (July) 1926.

8. Poeck, E.: Ueber Emphysembildung bei asphyktischen Neugeborenen, Monatschr. f. Geburtsh. u. Gynäk. **74:** 341, 1926.

9. DeCosta, E. J.: Spontaneous Pneumothorax of the Newborn Infant, Am. J. Obst. & Gynec. **39:** 578, 1940.

10. Fisher, J. H.: Spontaneous Pulmonic Interstitial and Mediastinal Emphysema in an Infant, Canad. M. A. J. **44:** 27, 1941.

11. Macklin, C. C.: Transport of Air Along Sheaths of Pulmonic Blood Vessels from Alveoli to Mediastinum: Clinical Implications, Arch. Int. Med. **64:** 913 (Nov.) 1939; Impediment to Circulation (Occasioned by Pulmonic Interstitial Emphysema and Pneumomediastinum), J. Michigan M. Soc. **39:** 756, 1940.

12. Fisher, J. H., and Macklin, C. C.: Pulmonic Interstitial and Mediastinal Emphysema, Am. J. Dis. Child. **60:** 102 (July) 1940.

13. Hamman, Louis: Spontaneous Mediastinal Emphysema, Bull. Johns Hopkins Hosp. **64:** 1 (Jan.) 1939.

14. Andrus, P. M.: A New Method for the Radiographic Exploration of the Mediastinum and Concealed Portions of the Pulmonic Fields, Radiology **23:** 97, 1934.

15. Complex procedures for the relief of mediastinal emphysema have been described but because of their unnecessary complexity would seem inadvisable. These may be found in:
Tregal, M.: Ein einfaches Verfahren zur Bekampfung des Mediastinalemphysems, Zentralbl. f. Chir. **38:** 420, 1911, and Furstenberg, A. C., and Yglesias, Louis: Mediastinitis: A Clinical Study with Practical Anatomic Considerations of the Neck and Mediastinum, Arch. Otolaryng. **25:** 539 (May) 1937.

contour, with apparent displacement of the upper part of the heart shadow and of the mediastinal structures to the right. There was an area of radiolucency just to the left of the mediastinum in the region of the second to third intercostal spaces, and there was some increased density in the upper part

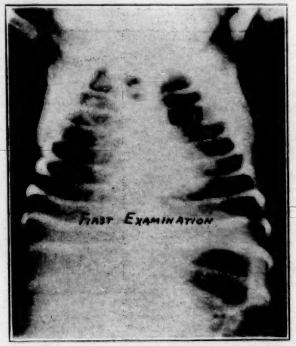


Fig. 1 (case 1).—Posterior-anterior view of the chest showing bilateral radiolucent shadows just lateral to the margins of the superior mediastinal density. These represent pools of air in the anterior mediastinal space. The cardiac shadow is suggestively abnormal in size and configuration. The heart and the superior mediastinal structures are partially displaced to the right. The upper areas of the lung fields have the appearance of either pulmonary congestion or patchy atelectasis, while the lower areas of the lung fields are hyperaerated.

of the right lung field. A lateral view (fig. 2) taken at the same time showed a distinct area of radiolucency beneath the sternum apparently displacing the heart and the mediastinal structures posteriorly. Study of the second film established the diagnosis of pneumomediastinum. During the succeeding twenty-four hours the condition of the infant became progressively worse, and a new roentgenogram showed some increase in the amount of mediastinal air. At this time the infant's

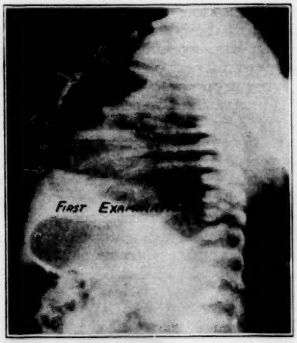


Fig. 2 (case 1).—Lateral view demonstrating sharply delineated shadows of encapsulated air in the anterior mediastinum just behind the posterior surface of the manubrium and the body of the sternum. Another globule of air is present in the region of the suprasternal notch. The shadows of the heart and the superior mediastinal structure are crowded and displaced posteriorly because of increased mediastinal pressure. Note the striking appearance in this view as compared with the posterior-anterior view (fig. 1).

condition seemed critical, and after consultation it was decided to attempt aspiration of the air.

A 21 gage needle attached to a 20 cc. Luer syringe was inserted into the third left interspace 1 cm. to the left of the sternal border and directed medially, parallel to the inferior surface of the sternum. There was a sudden expulsion of the plunger of the syringe to the 6 cc. mark, indicating the release of air under pressure. Within five minutes there was a dramatic change in the baby's condition. The color became pink, the respiratory rate dropped to about 100 per minute and the infant started to cry and to move about in his bed. A roentgenogram taken ten minutes after aspiration (fig. 3) showed that approximately 70 per cent of the air had been removed. Within three days the infant was clinically well, and roentgenograms showed complete disappearance of air.

The diagnosis in this case was made because lateral roentgen examination was done. As this procedure is not commonly carried out in the examination of the newborn it was felt that we might have been missing the diagnosis in a certain percentage of cases. Proof that we had been doing so was obtained when 3 additional cases were discovered during the next four months. In 2 of these the symptom was slight dyspnea. Enlargement of the thymus gland was suspected. The diagnosis of pneumomediastinum in both instances was obvious from lateral roentgenograms. In the third case there were moderate dyspnea and cyanosis, with some bulging of the precordium, absence of cardiac dulness and distant heart tones. The clinical impression of pneumomediastinum was substantiated by roentgenograms (figs. 4 and 5). Successive roentgenograms in this case were of great interest, as they showed an increasing mediastinal accumulation of air and finally spontaneous pneumothorax coincident with clinical improvement and evidence of reduction in mediastinal emphysema.

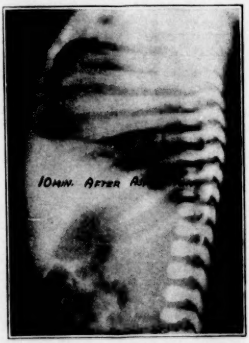


Fig. 3.—Lateral projection made immediately after therapeutic substernal aspiration. The air in the anterior mediastinum is now considerably diminished. Dramatic clinical improvement occurred within five minutes after aspiration.

It is interesting to speculate on the possibility that spontaneous pneumomediastinum may play a part in the production of types of spontaneous pneumothorax. One of us (B. G.) recently had an opportunity to observe a patient in the service of Dr. Henry G. Poncher, at the Cook County Hospital, Chicago, a 6 month old infant who had tuberculosis, in whom there developed pneumomediastinum and, subsequent to an increase in the amount of mediastinal air, spontaneous pneumothorax.

Davis and Stevens [16] reported that in 6 of 702 consecutive newborn infants the presence of spontaneous pneumothorax was proved by roentgenograms, an incidence of approximately 1 per cent. If pneumomediastinum was the precursor of pneumothorax in most instances one might expect an incidence of mediastinal emphysema of more than 1 per cent, as obviously pneumomediastinum does not lead to pneumothorax in all cases.

16. Davis, C. H., and Stevens, G. W.: Value of Routine Radiographic Examination of Newborn Based on Study of Seven Hundred and Two Consecutive Babies, Am. J. Obst. & Gynec. **20**: 73, 1930.

MECHANISM OF PRODUCTION

Spontaneous pneumomediastinum has been shown to result from pulmonary alveolar rupture (fig. 6) with subsequent passage of air through the interstitial tissue of the lung to the mediastinum. Macklin's [11] experimental work, done chiefly on cats, has demonstrated this mechanism conclusively. He showed that, when overdistention of a portion of the lung was artificially induced, alveolar rupture occurred, with the passage of air along the vascular sheaths to the mediastinum. It follows, therefore, that any condition which would tend to produce overdistention of a portion of the lung might result in mediastinal emphysema. The delicacy of structure of the lung of the newborn infant, and especially that of the premature infant, would seem to be a definite predisposing cause.

PATHOLOGIC CHANGES

Clinical and experimental work has shown that air in the mediastinum may extent upward to produce subcutaneous emphysema of the neck, may extent downward through the aortic hiatus to the retroperitoneal space and then, by rupture of the peritoneum produce pneumoperitoneum; or may rupture through the mediastinal pleura to produce pneumothorax (fig. 7). DeCosta [9] reported a case in which mediastinal emphysema, subcutaneous emphysema, pneumoperitoneum and pneumothorax all were present. In addition, a patient may have subcutaneous emphysema of the chest and the abdominal wall, interstitial emphysema of the uninvolved portions of the lung, pneumoprecordium or subcutaneous emphysema of the groin and thigh.

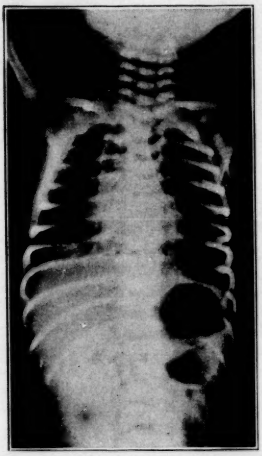


Fig. 4 (case 4).—Posterior-anterior projection. There is partial pneumothorax on the left complicating pneumomediastinum. Note the bilateral radiolucent shadows just lateral to the margins of the superior mediastinal density, representing residual mediastinal air.

If the air remains in the mediastinum and is under tension, various changes due to pressure on the surrounding structures may occur. The heart, great vessels, trachea, bronchi, lungs and vagosympathetic plexus all may be affected. Jehn and Nissen [17] have shown that no symptoms are evident until the intramediastinal pressure reaches zero. They found that as the pressure increases on the positive side changes occur rapidly and proceed as follows:

At first respirations become increased and shallow. Soon there is a drop in blood pressure. The pulmonary and systemic veins are the first affected, because of their elastic, easily compressed walls. By compression of the systemic veins the return flow of blood to the right auricle is diminished, and at the same time compression of the pulmonary veins causes diminution of the flow into the left auricle. There thus results congestion of the peripheral veins and of the lungs. The latter is an early sign and explains the dyspnea. The

Fig. 5 (case 4).—Lateral projection. There is encapsulated air in the anterior mediastinum substernally, and pneumomediastinum is present. Note the striking appearance as compared with the posterior-anterior view. Bulging of the precordium is clearly evident.

direct effect of compression on the trachea and bronchi and even the lungs themselves seems to be only a small factor in the production of dyspnea. The air in the mediastinum displaces the heart downward and posteriorly, so that the heart tones are apt to be distant and the percussion note over the heart tympanitic. Air over the heart may give rise to a peculiar crackling sound, synchronous with systolic contraction. This sound has been stressed as being of diagnostic value in the adult.[17]

From the foregoing discussion it would seem logical that many of the conditions mentioned might be ameliorated if spontaneous decompression of the mediastinum resulted with the formation of pneumothorax, subcutaneous emphysema or pneumoperitoneum. It also seems obvious that if spontaneous decompression did not occur and symptoms became alarming therapeutic decompression would be indicated.

SIGNS AND SYMPTOMS

Mediastinal emphysema in the newborn usually occurs during the first days of life. The condition manifests itself by respiratory and circulatory distress. Respirations become rapid and shallow, and cyanosis appears. Distention of the peripheral veins occurs, usually seen most easily in the neck. Heart tones are often distant, and one may hear a crackling sound synchronous with the heart beat. Occasionally one may note bulging of the precordium. Percussion fails to elicit cardiac dulness. If spontaneous rupture occurs one may feel subcutaneous emphysema or obtain signs of pneumothorax.

17. Jehn, W., and Nissen, R.: Pathologie und Klinik des Mestiastinalemphysems, Deutsche Ztschr. f. Chir. **206**: 221, 1927.

ROENTGEN DATA

Pneumomediastinum in the newborn may be suspected clinically, but the diagnosis is established definitely by roentgen examination.

The posterior-anterior view may reveal globules of air just outside what are usually considered the lateral margins of the superior mediastinum (figs. 1 and 4). The air shadows are extrapleural. If rupture through the mediastinal pleura occurs, pneumothorax becomes evident.

The lateral projection offers the most conclusive evidence and will in most instances prove to be the diagnostic film. In this projection one sees characteristically one or more pools of encapsulated air just beneath the shadow of the sternum. The density and the extent of the air shadow vary with the degree of emphysema (figs. 2, 3 and 5).

As air in the mediastinum gives rise to pressure phenomena, the lung fields may have the appearance of pulmonary congestion (fig. 1).

TREATMENT

The treatment should be conservative in most instances, oxygen and stimulants being administered as indicated. However, increasing dyspnea and cyanosis are an indication for therapeutic aspiration (case 1).

SUMMARY AND CONCLUSIONS

Spontaneous pneumomediastinum is characterized clinically by the sudden appearance of respiratory distress and varying degrees of cyanosis, with or without such signs as distention of the veins of the neck, bulging of the precordium, distant heart tones, obliteration of cardiac dulness, a crackling sound synchronous with the heart beat, subcutaneous emphysema or signs of pneumothorax.

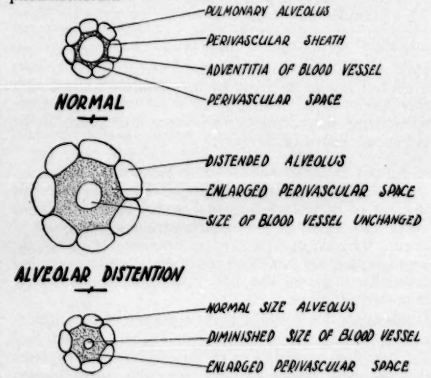


Fig. 6.—Theory of alveolar rupture according to Macklin.[11] The perivascular sheath is composed of the bases of pulmonary alveoli. Distention of the alveoli causes the perivascular sheath to be drawn away from the blood vessel, enlarging the perivascular space. Increasing pressure in the alveoli plus decreasing pressure in the perivascular space sets up a pressure gradient from the alveoli to the perivascular space and leads to rupture of alveolar bases. A similar pressure gradient results if there is a diminished blood flow to the lung, with consequent diminution in the size of the perivascular space. (This probably explains the high incidence of spontaneous pneumothorax among infants with congenital heart disease.)

The diagnosis is established with the aid of roentgenograms of the chest. The pathognomonic appearance of the lateral view is stressed.

Macklin's experimental work suggested that the cause of spontaneous pneumomediastinum is overdistention of the lung and demonstrated the path of air from ruptured alveoli along the vascular sheaths to the mediastinum.

It appears likely from experimental and clinical observation that pneumomediastinum may secondarily cause pneumothorax, pneumoperitoneum or subcutaneous emphysema.

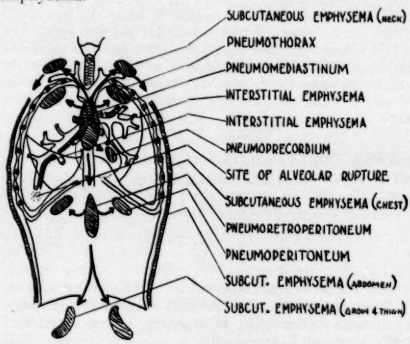


Fig. 7.—The dotted area in the lower part of the right lung field represents an area of alveolar rupture. The shaded areas represent air in abnormal situations. Note that air from the mediastinum may enter the uninvolved portion of the lung and produce interstitial emphysema. The remainder of the diagram is self explanatory and shows the direction of the spread of air. Pneumothorax occurs if there is rupture of the mediastinal pleura, and pneumoperitoneum occurs if there is rupture of the peritoneum.

Treatment is usually conservative, but increasing dyspnea and cyanosis are an indication for therapeutic aspiration.

It is felt that pneumomediastinum in the newborn is a definite clinical entity and that its recognition and treatment, as described in the case reported, should result in a decrease in the neonatal mortality.

2501 Devon Avenue.

ABSTRACT OF DISCUSSION

DR. C. C. MACKLIN, London, Canada: In October 1940 I wrote, regarding pulmonic interstitial emphysema and pneumomediastinum, that "prompt appropriate therapy may save many lives, especially in children," and I feel that Dr. Gumbiner's experience supports this prediction. I am gratified at that accurate diagnosis made in these cases, and particularly for the brilliant demonstration by appropriate x-ray studies. Most of all I am grateful for this demonstration of a clinician who is on the lookout for cases of this sort; for without that attitude and scientific curiosity there can be no progress in this field. I feel assured that aberrant air in the interstitial tissue of the lungs and mediastinum, and contiguous parts, constitutes the basis of a definite clinical entity which has gone undiagnosed in most instances. I have spoken of this clinical entity as "air block," because the air presses on the pulmonary arteries and veins and on the heart and great vessels, so impeding the circulation and giving rise to cyanosis and dyspnea. Air block occurs much more frequently than is realized, and it probably occurs at all ages and under a variety of conditions. Intensive research will give it greater prominence as a serious clinical condition and as a cause of death. Often, I feel, it is associated with bronchopneumonia, and thus its manifestations may be masked. The authors have rendered a real service to the public and medical profession in making known the results of their careful studies. It is a field in which but little work has been done heretofore. That these newborn pneumonopathy cases may end fatally is shown by the experience of John H. Fisher, who has had two cases come to autopsy, one of which has been reported (*Canad. M. A. J.* **44**:27 [Jan.] 1941). In

this the pathologic findings are like those of the experimental animals which I have studied. I feel that atelectasis is an important predisposing cause, since it is associated with a compensatory emphysema. It is through the weakened bases of the alveoli which overlie the smaller branches of the pulmonary arteries and veins that the air breaks through into the connective tissue around these vessels, and, continuing to leak into these sheaths, it makes its way along them to the mediastinum. As the air accumulates it presses on the vascular system, so impeding the circulation and possibly giving rise to pain as well. In these infants it seems reasonable to assume that there was failure of expansion in certain areas of the lung, with compensatory alveolar ectasia, and that air broke through these stretched alveolar bases, probably during fits of crying.

PROCTOLOGY: ITS QUALIFICATIONS AND PROGRESS

CHAIRMAN'S ADDRESS

FRANK C. YEOMANS, M.D.
NEW YORK

Near the end of the last century a national association in gastroenterology and another in proctology were organized. Since then both groups have functioned continuously and successfully.

In 1916, mainly through the persistent efforts of leaders of these associations, the American Medical Association officially recognized both specialties by establishing the Section on Gastro-Enterology and Proctology. Today we meet in the twenty-fifth annual session. The eminent success of the section has fully justified its creation. Through the untiring efforts of a qualified personnel under zealous leaders these special fields of medicine have developed progressively and are now established on a sound scientific basis.

Briefly, the primary functions of any medical specialty are:

1. Scientific. The development of the specialty itself by scientific research and investigation and clinical observation.

2. Educational. (*a*) Teaching its elemental principles to undergraduate and graduate students, (*b*) the training of competent young doctors who contemplate taking up the specialty and (*c*) dissemination within the profession, by the written and spoken word, of useful knowledge which can be safely and effectively applied to practice.

3. Service. Supplying the highest type of medical service to the patient, which after all is the chief objective of medicine.

This program places a heavy but just responsibility on every association of specialists.

Our two special branches are concerned with the alimentary tract and its associated apparatus. Gastroenterology is essentially medical while proctology is chiefly surgical.

Speaking particularly for proctology, what qualifications should those possess who would enter this field, which may be defined as that of the bowel distal to the ileocecal valve.

To qualify properly in proctology one should enter the specialty through the door of general surgery. The essential personal qualifications for any honest specialist worthy of the name are the three I's: Intelligence, Industry, Integrity.

Intelligence and scientific attainment may be gaged in fair degree by personal interview and examinations, for example those required for fellowship in the American College of Surgeons or for diplomate in surgery.

Integrity, however, is a moral quality which transcends all others. Without integrity the patient may be exploited and the specialty discredited.

It is generally recognized that many more adequately trained, ethical proctologists are needed in many sections of the country. The two agencies that can best solve the problem are the medical schools and the general hospital.

The curriculums of the undergraduate medical schools are sorely taxed. Yet, somewhere in this course each student should have the opportunity of seeing, under the guidance of a competent instructor, the common lesions of the distal portion of the bowel, of doing digital palpations and instrumental inspection of the rectum and pelvic colon and be taught that these procedures should, as a rule, precede roentgen studies.

The surgical staff of the general hospital should encourage a qualified proctologist to organize an outpatient clinic, follow up patients admitted to the wards, have access to the laboratories, and, when he has demonstrated his competence, be permitted to operate in both minor and major conditions within his field. If this reasonable plan were generally adopted, the interns would receive instruction sufficient to avoid the frequent glaring errors in diagnosis and treatment of proctologic conditions after they have finished their hospital residence.

Gastroenterology and proctology are the only clinical specialties represented among the sections of the American Medical Association which do not have independent boards for certification.

The Council on Medical Education and Hospitals has recently approved the action of the American Board of Surgery in regard to the recognition of proctologists. This provides that applicants must first meet the same requirements and pass the same examinations as applicants for certification in general surgery. In addition, applicants for proctologic surgery are required to pass a clinical test to be given by a committee representing this special field. We urge all those who contemplate specializing in proctology to become diplomates of the American Board of Surgery.

PROGRESS IN PROCTOLOGY

Medicine is not static but progressive. The scientific method of advance is usually slow, seldom dramatic, yet a brief survey of some of the advances in proctology during the past quarter century demonstrates that substantial progress has been made and that opinion has crystallized as to the best management of several important diseases.

Anatomy.—A restudy of the anatomy of the anorectum by Milligan and Morgan,[1] and others has clarified the complex musculature of this region and explains the channels through which infection spreads—two important and practical contributions to an understanding of the morbid anatomy of the parts and the surgical procedure indicated for their correction.

Proctosigmoidoscopy.—At long last the value of proctosigmoidoscopy is generally recognized. It is a routine in well equipped clinics but, alas, many hospitals still do not possess suitable equipment or have trained personnel, and all too frequently the procedure is omitted by the general practitioner. The modern

Read before the Section on Gastro-Enterology and Proctology at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 5, 1941

1. Milligan, E. T. C.; Morgan, C. N.; Jones, L. E., and Officer, R.: Surgical Anatomy of the Anal Canal and the Operative Treatment of Hemorrhoids, Lancet **2**: 1119 (Nov. 13) 1937.

pneumoelectric tube is invaluable in diagnosis (including biopsy), for check-up observation and for treatment (*a*) with topical applications, (*b*) for implanting seeds of radon and (*c*) for surgical diathermy of tumors by fulguration, coagulation or the electric snare.

Roentgenologists as well as clinicians agree that proctosigmoidoscopy should precede a roentgen study of patients having symptoms referable to the distal portion of the bowel because the rectum and the pelvic colon, accessible to palpation and direct inspection, are the sites of about 75 per cent of all tumors of the large bowel. The roentgen ray frequently misses a small intestinal growth within the bony pelvic girdle while it is still small and amenable to early treatment. Moreover, these segments are involved in nearly all inflammatory and infectious conditions of the large bowel. In fact the disease process begins in the rectum in the majority of cases of amebic and bacillary dysentery and chronic ulcerative colitis.

Of course a roentgen study of the remaining colon or entire gastrointestinal tract is essential in many instances. Refinements of roentgenologic technic now frequently show the mucosal pattern and reveal many lesions which were formerly missed. Sigmoidoscopy and a roentgen study are not opposed to each other; on the contrary, they complement each other.

Anesthesia.—Aside from local infiltration anesthesia, caudal anesthesia, sacral-block anesthesia, spinal anesthesia and anesthesia with nitrous oxide-oxygen, ether, cyclopropane, avertin with amylene hydrate and pentothal sodium all have their indications in rectal surgery, and selection should be made appropriate to the individual patient.

Benacol,[2] the pioneer oil-soluble anesthetic, was originated in my clinic in 1927.[3] It stimulated the production of several other similar combinations of chemicals in solution in oil. They are useful for the prolonged relief of pain in acute lesions, notably fissure. The relaxation of the sphincter muscles which follows the proper injection of an oil-soluble anesthetic enables the surgeon to dilate the muscles painlessly, exposing the lesion for topical applications, and thus effect a cure in the large majority of acute fissures of the anus.

Likewise, the injection of an oil-soluble anesthetic immediately after an operation for cryptitis, fissure or hemorrhoids gives in most instances postoperative comfort lasting from a few days to a week.

Pruritus Ani.—In my experience the injection of an oil-soluble anesthetic has afforded temporary or permanent relief in approximately 70 per cent of the many cases of so-called idiopathic pruritus ani in which it has been used.

Hemorrhoids.—Since it has been placed on an ethical and sound scientific basis, the injection treatment of internal hemorrhoids with sclerosing solutions has grown in popularity. The two agents used are a 5 per cent aqueous solution of quinine and urea and a 10 per cent aqueous solution of phenol or 5 per cent phenol in a vegetable oil.

The injection method yields excellent palliation in about 85 per cent of cases of uncomplicated internal hemorrhoids which comprise approximately 50 per cent of all cases. Relief is permanent in many cases, but recurrence in from one to five years in approximately 30 per cent requires another course of injections. However, one should remember if the patient has no general condition which bars surgery that hemorrhoidectomy is the procedure of choice for the permanent relief of all types of hemorrhoids.

Fistula.—Infection, usually of the anal crypts, is generally recognized as the basis of fissure, abscess and fistula. The latter is usually excised rather than incised, unless the internal opening is so high in the rectum that excision would impair control. In this event, operation in stages is indicated. If feasible, a tuberculous fistula should be excised *en masse* and wide of the disease, preferably with electrosurgery rather than with the scalpel. This technic seals the lymphatics and thus prevents the spread of a tuberculous infection.

Chronic Ulcerative Colitis.—This is still a puzzling problem. More knowledge of food, of nutrition, of the biologic processes and especially of the vitamins may shed light on the obscurity. Chemotherapy is achieving dramatic results in various types of infection. The sulfonamides have proved their worth in many cases of chronic ulcerative colitis. These experiences give promise that a more effective therapy than is now available will be developed for this serious disease entity.

The possibility of amebiasis should be ruled out in every case of nonspecific chronic ulcerative colitis by a careful sigmoidoscopy which reveals the pathognomonic lesions in early cases and frequently establishes the correct diagnosis, although the laboratory examination of the stools is negative.

Joseph Felsen, after extensive clinical observation and laboratory research, has emphasized the role of bacillary dysentery in the causation of chronic ulcerative colitis. To date, this theory has not received wide acceptance.

Stricture.—A wholly new concept of the etiology of inflammatory stricture of the rectum has arisen from the work of Durand, Nicholas and Favre in 1913[4] and of Frei in 1925.[5] Syphilis and gonorrhea have been relegated to a minor role. Lymphogranuloma venereum is generally conceded to be the etiologic factor in the vast majority of cases. In practically all cases of stricture with proctitis, the diagnosis of lymphogranuloma is established by obtaining a positive cutaneous reaction to an intradermal injection of an inactivated antigen. Frei used human antigen (1925). Grace and Suskind[6] introduced mouse brain antigen (1934), and recently (1940) Rake, McKee and Shaffer[7] have provided lygranum antigen, produced by growing the virus in the yolk sac of the developing chick embryo. Mouse brain antigen and lygranum are comparatively free from the disadvantages of the human product, yet they appear to be at least equal to the Frei test in sensitivity and specificity.

Chemotherapy with the sulfonamides is proving to be a sovereign remedy for control of lymphogranuloma venereum and for prevention of stricture formation. Unfortunately, in most cases the disease has already progressed to the stage of stricture before coming under treatment. Nevertheless, the sulfonamides should be administered for their virostatic effect on the disease, although they exert little or no change in the stricture

2. Benacol contains benzocaine 3 per cent, procaine 2 per cent and benzyl alcohol 5 per cent in sweet almond oil.

3. Yeomans, F. C.; Gorsch, R. V., and Mathesheimer, J. L.: Tr. Am. Proctol. Soc., 1927.

4. Durand, M.; Nicholas, Joseph, and Favre, M.: Lymphogranulomatose inguinale subaiguë d'origine génitale probable, peutêtre Vénérienne, Bull. et mém. Soc. méd. d. hôp. de Paris **35**: 274, 1913.

5. Frei, Wilhelm: Eine neue Hautreaktion bie Lymphogranuloma inguinale, Klin. Wchnschr. **4**: 2148 (Nov. 5) 1925.

6. Grace, A. W., and Suskind, F. H.: Successive Transmission of Virus of Lymphogranuloma Inguinale Through White Mice, Proc. Soc. Exper. Biol. & Med. **32**: 71 (Oct.) 1934.

7. Rake, Geoffrey; McKee, Clara M., and Shaffer, M. F.: Agent of Lymphogranuloma Venereum in the Yolk-Sac of the Developing Chick Embryo, Proc. Soc. Exper. Biol. & Med. **43**: 332 (Feb.) 1940.

itself. Usually the latter can be managed successfully with irrigations of potassium permanganate and biweekly dilation with Wales soft rubber bougies, in progression to number 7.

Resistant cases may require internal longitudinal incision of the stricture at three points (right, left and posterior) with diathermy, to be followed by routine dilation and irrigations. Multiple fistulas occurring with esthiomene or elephantiasis may require colostomy for relief. Rarely a stenosing tubular rectitis is best treated by perineal proctectomy. I did a perineal excision of the rectum on 2 patients having extensive strictures, intractable to other forms of treatment. One was a Negro woman aged 31, the other a white man aged 49. The result was excellent in both instances.

Carcinoma.—The majority of experienced surgeons and many pathologists now believe that the bulk of adenocarcinomas of the large bowel are mediated through adenomas and that their early detection and destruction is the surest measure for the prevention of cancer. Among the many adenomas of the lower part of the bowel which I have removed there were 18 specimens that showed precancerous changes or definite cancer. If the tumor is pedunculated, it is most simply and thoroughly removed with the high frequency electric snare; if sessile, it is destroyed by fulguration or coagulation.

Although definite palliation and a few five year cures have been reported of carcinoma of the rectum treated with irradiation, the results in general have been disappointing. One of the chief reasons for this failure is that the mucosa of the bowel is highly sensitive to the rays while the cancer is very resistant. Moreover, the unfavorable systemic effect of the roentgen rays, especially on the blood and blood-forming organs, tends to lower the vital resistance. As a consequence, the clinical consensus is that surgical excision is the best procedure in all operable cases, that is, if the patient's general condition is such that he can bear a major operation or he can be rehabilitated; second, that the lesion itself is so limited that there is reasonable expectation that it can be completely removed.

Extensive experience with the one stage abdominoperineal excision of Miles, with permanent colostomy, yields by far the greater number of five year cures than any other for cancer of the rectosigmoid, lower pelvic colon and rectal ampulla. This rectosigmoid segment is the site of about two thirds of all cancers of the sigmoid and rectum. The operative mortality is within 15 per cent in the hands of surgeons competent in this field but is much higher for the average surgeon who sees comparatively few cases. The moral is that some one surgeon on a hospital staff who is especially interested should have the care of these patients so that he may perfect his technic.

Carcinoma situated in the sigmoid 1 or more inches above the pelvic pouch may be resected safely by inverting the distal end and permanent colostomy, more rarely by an exteriorization procedure and later closure of the colostomy to reestablish continuity.

For a rectal cancer, the upper limit of which can be reached by the finger, perineal excision gives excellent results and a very low operative mortality. It is especially indicated in the treatment of obese subjects, the debilitated and the aged.

This brief survey of progress during the last twenty-five years augurs well for the future of proctology.

555 Park Avenue.

THE TREATMENT OF MODERATELY SEVERE SCARLET FEVER

A STUDY OF ALTERNATE PATIENTS TREATED WITH SULFANILAMIDE, CONVALESCENT SERUM AND SCARLET FEVER ANTITOXIN

F. H. TOP, M.D.
AND
D. C. YOUNG, M.D.
DETROIT

The value of scarlet fever convalescent serum and scarlet fever antitoxin in the treatment of moderately severe scarlet fever was demonstrated by a control study reported in a previous paper.[1] Early in 1937 sulfanilamide was introduced in this country as a therapeutic agent for the treatment of hemolytic streptococcus infections, and a similar therapeutic study including sulfanilamide was repeated. During the period Aug. 1, 1937 through July 31, 1938 sulfanilamide, convalescent serum and antitoxin were given to alternate patients who on admission to the Herman Kiefer Hospital showed scarlet fever of a moderately severe grade. As in the previous study, the determination of the severity of the condition and the necessity for a therapeutic agent was the responsibility of two resident physicians who had been in the service for more than ten years. They had no part in the choice of therapeutic agent to be used. Whenever antitoxin was in order a serum sensitivity test was performed; if positive, either convalescent serum or sulfanilamide was used and the patient excluded from the series, and the next patient with scarlet fever of a moderately severe type was given antitoxin if the serum sensitivity test was negative. Both convalescent serum and antitoxin were administered in the hospital admitting room, while sulfanilamide was given by mouth as soon as the patient reached the pavilion, a period not exceeding one hour after admission to the hospital.

The sulfanilamide used was obtained in the form of tablets during the early part of the study and later was furnished in powdered form. The doses administered initially and subsequently at four hour intervals, called respectively the initial dose and the maintenance dose, were chosen rather arbitrarily, for there was little information regarding dosage when this study was begun. The dosage shown in table 1 is based on weight and shows both the initial dose and the maintenance dose. At present an initial dose larger than the maintenance dose is unnecessary for infections which are not severe, but during the time that the study was in progress a large initial dose was administered. The patient's weight was roughly estimated and the prescribed dose for that weight was given. The average total dose for all patients treated with sulfanilamide was 12.4 Gm., and the average duration of therapy was six and three-tenths days. The weight of the patient had no influence on the duration of therapy. Sulfanilamide determinations were not determined as a routine because the amount of drug given was too small to warrant its use. In the instances in which the reading was determined, none were found to be above 5 mg. per hundred cubic centimeters of blood.

This study was aided in part by WPA Project 82-4-120.
The sulfanilamide used in this study was furnished by Merck & Co., Inc.

From the Communicable Disease Service, Herman Kiefer Hospital; the Detroit Department of Health, and Wayne University College of Medicine.

1. Top, F. H., and Young, D. C.: Specific and Nonspecific Serum Treatment of Scarlet Fever, Am. J. Pub. Health **29**: 443 (May) 1939.

The scarlet fever convalescent serum used was obtained from donors who had recovered from the disease within one year of their being bled in the serum clinic of the department of health. The various batches of serum were not tested for antitoxin content, but it is likely that the amount of scarlet fever antitoxin present in each cubic centimeter of serum was not greater than the amount (less than 2 units per cubic centimeter) determined by testing thirty-three batches of serum used in the previous study. Convalescent serum was given intramuscularly, and the dose was 30 cc.

Scarlet fever antitoxin was obtained from the laboratories of the state department of health, and one therapeutic dose of serum (6,000 units) was administered by the intramuscular route.

The therapeutic agents used in this study were given to alternate patients after they were admitted to the Communicable Disease Service. During the year 390 cases of moderately severe scarlet fever were divided equally among the three therapeutic agents used. The method by which the agents were assigned was considered paramount to obtaining comparable groups. The following characteristics, which should be similar for all treatment groups, were tested for statistical comparability by using the chi square test: distribution of cases by month, by age, by temperature on admission to the hospital, by the day of disease on which treatment was begun and by the proportion of cases in which there were significant associated conditions. The three treatment groups did not differ with respect to the characteristics enumerated and for the purpose of this study may be looked on as similar in all respects except the kind of treatment received.

TABLE 1.—*Dosage of Sulfanilamide*

Body Weight, Pounds	Initial Dose, Grains	Maintenance Dose, Grains
20- 30	10	2½
30- 50	15	5
50- 70	20	5
70- 90	25	5
90-110	30	10
110-130	35	10
130+	40	10

TABLE 2.—*Number and Proportion of Cases with Augmented Treatment by Type of Therapy*

Therapy	Total Cases	Augmented Treatment	Per Cent Augmented Treatment
Sulfanilamide	130	11	8.5
Convalescent serum	130	5	3.8
Antitoxin	130	10	7.7

RESULTS OF TREATMENT

The therapeutic effect of the agents utilized was tested by observing the result with respect to (1) the number of patients who needed subsequent therapy similar to that used in the study, (2) the decline in temperature, (3) the number of patients with complications, (4) the occurrence of complications, (5) the proportion of patients with multiple complications, and (6) the proportion of patients dismissed from the hospital at the end of the minimum isolation period. Duration of the rash was not estimated because it is difficult to evaluate unless based solely on the presence or absence of rash.

AUGMENTED TREATMENT

For purposes of this study only one dose of scarlet fever convalescent serum (30 cc.) and one dose of scarlet fever antitoxin (one therapeutic dose) were administered by the intramuscular route, while the dose of sulfanilamide was governed by weight. It is common clinical experience to find that some patients with moderately severe scarlet fever need additional therapy. Occasionally, twenty-four hours after admission, the temperature has remained the same as on admission or has risen or the patient's general condition has shown no improvement. Whenever additional therapy was necessary, this was considered as augmented treatment, was so recorded and the case excluded from the study. The number and proportion of patients needing augmented treatment is shown in table 2. It will be observed that but few patients in each treatment group were given additional therapeutic aid. In the convalescent serum group it was necessary for only 5 patients, and the number was 10 and 11 for the antitoxin and sulfanilamide groups respectively. Convalescent serum appears to advantage, but the differences noted and not significant for a study of this size ($p = 0.29$).[2] Patients needing augmented treatment were excluded from further consideration in this study, causing slight changes in the totals of the treatment groups.

TABLE 3.—*Temperature Pattern by Type of Therapy*

Temperature Pattern	Sulfanilamide		Convalescent Serum		Antitoxin	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Normal	71	59.7	79	63.2	90	75.0
Lysis (5 days)	28	23.5	21	16.8	12	10.0
Continued fever	20	16.8	25	20.0	18	15.0
Total	119	100.0	125	100.0	120	100.0

TABLE 4.—*Complicated Cases by Type of Therapy*

Therapy	Total Cases	Number Complicated	Per Cent Complicated
Sulfanilamide	119	55	46.2
Convalescent serum	125	63	51.2
Antitoxin	120	61	50.8

TABLE 5.—*Occurrence of Complications by Type of Therapy*

Complication	Sulfanilamide		Convalescent Serum		Antitoxin	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Rhinitis	31	26.1	41	32.8	47	39.2
Cervical adenitis, nonsuppurative	16	13.4	15	12.0	15	12.5
Otitis media, catarrhal	9	7.6	7	5.6	..	....
Otitis media, suppurative	8	6.7	9	7.2	7	5.8
Mastoiditis	2	1.7	2	1.6	1	0.8
Bronchitis	7	5.9	4	3.2	6	5.0
Arthritis	4	3.4	3	2.4	..	...
Paronychia	3	2.5	3	2.4	8	6.7
Abscess, soft parts	..	...	4	3.2	5	4.2
Nephritis	1	0.8	2	1.6	2	1.7
Bacteremia	..	...	1	0.8	..	...
Carditis	..	...	1	0.8	..	...
Ethmoiditis	..	...	..	...	1	0.8
Bronchopneumonia	..	...	..	...	1	0.8
Total cases treated	119		125		120	

2. When p is 0.03 or below, differences found are generally considered significant; that is, they are not considered to be due to chance variations.

TEMPERATURE PATTERN

The therapeutic response, as evidenced by an effect on temperature, is shown in table 3. The patients were divided into three groups with reference to temperature: the first, a group in which fever declined to normal or within one degree of normal within twenty-four hours; the second, a group in which fever declined by lysis within a period of five days, a result ordinarily noted in patients with moderate scarlet fever not treated with serum; third, a group in which fever was maintained or in which a temporary drop occurred, followed by a rise within twenty-four hours.

A temperature decline to normal occurred in 75 per cent of patients given antitoxin, in 63.2 per cent given convalescent serum and in 59.7 per cent given sulfanilamide. Among the remaining patients in each treatment group a decline by lysis occurred most frequently among the sulfanilamide-treated group, followed in order by the convalescent serum and the antitoxin-treated group, the respective proportions of each treatment group being 23.5, 16.8 and 10 per cent. Continued fever occurred less commonly among patients treated with antitoxin and sulfanilamide, while convalescent serum showed the poorest results in this respect. The differences noted appear to be significant and statistically are borderline ($p = 0.04$). A larger series of cases might demonstrate real differences. However, variation between antitoxin and convalescent serum is not appreciable and bears out past experience.

COMPLICATED CASES

Complications occur in moderately severe scarlet fever more frequently than one would surmise, and this is particularly true of hospitalized cases. This would not be so evident if only major complications, such as otitis media, mastoiditis, cervical adenitis and other less frequent but equally severe complications were solely included. In this analysis all complications incident to scarlet fever infection are considered, even such mild conditions as serous rhinitis and paronychia. Table 4 shows the number of complications in each of the treatment groups, and it is readily apparent that the proportion of complicated cases in the three groups is similar. The advantage shown by sulfanilamide is very slight and not significant.

OCCURRENCE OF COMPLICATIONS

The commonest complications of scarlet fever encountered in hospital practice are, in order of their frequency, rhinitis, cervical adenitis and otitis media. This is borne out in this study by the data in table 5. Rhinitis does not occur with equal frequency in the three treatment groups. The proportion of patients with rhinitis is lowest in the sulfanilamide treated group (26.1), the proportion in the antitoxin group is highest (39.2), while the convalescent serum group is midway between (32.8). The differences are not significant ($p = 0.09$). No real differences are noted for nonsuppurative cervical adenitis. The number of times that catarrhal or suppurative otitis media developed was small for the entire experience. Catarrhal otitis media occurred with the same frequency in the sulfanilamide and convalescent serum groups but did not occur in the antitoxin group. With respect to suppurative otitis media, the proportion is about the same for all treatment groups. When catarrhal and suppurative otitis media are combined, it is apparent that there is a difference in favor of the antitoxin group, but this is not statistically significant ($p = 0.08$).

The remainder of the complications listed in the table occur too infrequently to warrant comment. Consideration of the entire table must lead to the conclusion that variations in the treatment groups are not striking (table 5).

MULTIPLE COMPLICATIONS

The number of complications per person is shown in table 6. As might be expected with the mild form of scarlet fever now encountered, few persons had more than two complications each. The proportion of complications in each group is about the same, and differences are due to chance variation ($p = 0.84$).

The table also shows the number of complications per person with complication, which is the same for each treatment group. The number of complications is less than 1 per treated patient and is similar for each treatment group (table 6).

TABLE 6.—*Number of Complications per Person by Type of Treatment*

Treatment	Number of Complications per Person				Complications Group			Entire Group	
	1	2	3	4	Number of Persons	Number of Complications	Complications per Person	Number of Persons	Complications per Person
Sulfanilamide									
Number	35	14	6	..	55	81	1.5	119	0.7
Per cent	63.6	25.5	10.9						
Convalescent serum									
Number	42	14	6	1	63	92	1.5	125	0.7
Per cent	66.7	22.2	9.5	1.6					
Antitoxin									
Number	36	19	5	1	61	93	1.5	120	0.8
Per cent	59.0	31.1	8.2	1.6					

DISMISSAL STATUS

As previously noted,[1] the dismissal status is not a particularly good criterion for evaluation of treatment, especially if only one dose of serum is administered. Both antitoxin and convalescent serum cannot reasonably be expected to exert much influence on the incidence of complications which occur two weeks after the administration of serum. The same is true in the

TABLE 7.—*Dismissal Status of Cases Admitted to the Hospital*

Age	Total in Group			Late Dismissals			Proportion of Late Dismissals		
	S	C	A*	S	C	A	S	C	A
Under 1 year									
1- 4 years	27	30	37	12	12	21	44.4	40.0	56.8
5- 9 years	57	47	52	19	18	8	33.3	38.3	15.4
10-14 years	21	22	18	5	3	5	23.8	13.6	27.8
15-19 years	6	11	4	—	3	—	....	27.3	....
20+ years	8	15	9	—	1	1	....	6.7	(11.1)†
Totals	119	125	120	36	37	35	30.3	29.6	29.2

* S = sulfanilamide, C = convalescent serum and A = antitoxin.
† ( ) = a percentage based on less than 10 cases.

instance of sulfanilamide, the administration of which was maintained only during the first week in the hospital. An influence might be exerted on the incidence of complications during the first two weeks of illness, but a large proportion of complications occur during and after the third week of convalescence. However, the soil may be prepared, so to speak, during the first two weeks for what the patient may be exposed to in the hospital during the remainder of his stay. For this reason, data on the dismissal status are included. In the previous study, no difference was noted in the proportion of patients dismissed at the end of the required minimum period of isolation, and in this clinical experiment the proportion of late dismissals in each of the treatment groups (total cases) is practically the same. There are striking differences observed when age is considered, but too much weight cannot be given to the results because of small numbers.

COMMENT

Although the clinical study was completed in the late summer of 1938 and the analysis early in 1939, there was some reluctance on our part to publish the results. Timidity was due not to lack of confidence in the validity of the data or in the manner of collection but rather to the fact that in our hands sulfanilamide appeared to be only slightly less valuable in the dosage given than one therapeutic dose of antitoxin (6,000 units) or 30 cc. of convalescent serum administered intramuscularly. By this time it was already evident experimentally that sulfanilamide did not affect the clinical manifestations or complications directly caused by toxin elaborated by hemolytic streptococci.

In recent years scarlet fever has become a progressively milder disease. Both septic and toxic attacks still occur, but the number is proportionately smaller. The therapeutic agents used in this study would be inadequate in amount for septic and toxic attacks. Moderately severe scarlet fever also occurs less commonly, as shown by the fact that this type represented 37.8 per cent of 10,666 cases of scarlet fever dismissed from the Herman Kiefer Hospital during the five year period 1927-1931, while during the five year period 1932-1936 the proportion of this type among 10,777 dismissals was 21.7 per cent, a considerable decline. Both the organism and its toxin contribute to the clinical syndrome called scarlet fever, but the quantitative effect of each in any given case would appear at best controversial. Antitoxin is valuable in the treatment of moderately severe scarlet fever and is particularly indicated for toxic manifestations. It has been amply demonstrated that scarlet fever convalescent serum is of value in the treatment of moderately severe scarlet fever. Soon after sulfanilamide was used in the treatment of streptococcic infections it was found that it had little effect on toxic manifestations of streptococcus origin. This finding was borne out in early reports. Any value that sulfanilamide might have in the treatment of scarlet fever would likely be due to antibacterial properties, and it is now generally conceded that the effect is bacteriostatic rather than bactericidal.

During the past three years, there have been numerous reports dealing with the use of sulfanilamide in the treatment of scarlet fever. These clinical reports fall into two groups; first, those which include scarlet fever among other streptococcic infections treated and, secondly, those which deal solely with scarlet fever. The first group does not lend itself to evaluation, while the second group includes few studies which are adequately controlled. The use of sulfanilamide in the treatment of scarlet fever has been reviewed sufficiently often to allow its omission in this paper. We are interested in the fact that there is a difference of opinion concerning the value of sulfanilamide in moderate or moderately severe scarlet fever.

Of all articles published thus far, that of French [3] appears on inspection to be the best planned and the most exact in the method of assignment of cases to treatment groups. She finds that the use of sulfanilamide is not justified in the treatment of scarlet fever. The dose used was large and therapy was maintained for the entire period of isolation. However, when indicated, she gave scarlet fever antitoxin on admission to any patient who was thought to require it, irrespective of whether the patient was to receive sulfanilamide or not. Although the number receiving antitoxin in each group was the same, we feel that any therapeutic agent to be tested should be given alone and not in conjunction with another agent, particularly one of such proved value as scarlet fever antitoxin. For this reason we excluded patients with augmented treatment from further consideration in the series. French probably labored under the same difficulty that we did, namely the undesirability (in the eyes of the medical and nursing personnel) of withholding a known valuable therapeutic agent from one half of the cases (controls). We partially circumvented this problem in the previous study by introducing as an unknown serum diphtheria antitoxin one dose of which contained 1,000 units, alternating this with scarlet fever convalescent serum and antitoxin.

In the present study sulfanilamide replaced the diphtheria antitoxin and we feel that, with each patient in the study receiving but one therapeutic agent instead of two, the exact effect of any one can better be determined. For this reason the present study is submitted. Additional studies are needed to confirm or refute the findings because clinical evaluation cannot feasibly be based on deaths but must rely on differences in behavior in cases in which recovery occurred and this entails a few large studies or many well conducted small ones. When results of therapy are so nearly alike, as noted in this study, it is impossible to state that any of the therapeutic agents used are of value, unless the impression is based on other clinical or experimental studies. In the previous study 30 cc. of convalescent serum and one therapeutic dose of antitoxin (6,000 units) given intramuscularly were found to be of equal value in the treatment of moderately severe scarlet fever. The same statement applies in this study.

SUMMARY AND CONCLUSIONS

1. During the one year period Aug. 1, 1937 through July 31, 1938 a series of 390 patients with moderately severe scarlet fever were treated under control conditions with (*a*) sulfanilamide, (*b*) scarlet fever convalescent serum and (*c*) scarlet fever antitoxin.

2. Status-on-admission factors known to influence the course of the disease, such as (*a*) the month of attack, (*b*) age, (*c*) temperature on admission, (*d*) day of disease on which treatment was begun and (*e*) proportion of cases presenting significant associated conditions, were similar and not significantly different for the three treatment groups.

3. French, Jane O.: The Sulfanilamide Treatment of Scarlet Fever, J. Hyg. 39: 581 (Sept.) 1939.

3. The results of this study indicate that there is no great difference in the therapeutic effect in cases of moderately severe scarlet fever when sulfanilamide, convalescent serum and antitoxin are administered in the dosage used. Antitoxin and convalescent serum give better results than sulfanilamide, but the differences are not statistically significant.

4. In a previous study, 30 cc. of scarlet fever convalescent serum and one therapeutic dose of scarlet fever antitoxin (6,000 units) gave equally good results when administered by the intramuscular route. Their similarity in value in the dosage cited is again demonstrated.

5. No attempt is made to explain the results obtained. The study is presented as an addition to the literature on the subject.

Taylor and Hamilton Avenues.

HEALTH EDUCATION

AN APPRAISAL

DONALD B. ARMSTRONG, M.D., Sc.D.
Third Vice President, Metropolitan Life Insurance Company
NEW YORK

Perhaps the time has come, as is apt to be the case periodically in most fields, for a fresh orientation as to what in general we mean by health education. There are still those in public health who see little or no value or scope in health education. There are also those, in increasing numbers, who appear to think that health education and public health are practically synonymous terms—coextensive in range and content. Between these two extreme views, what is health education's real status?

Parenthetically, let me make it clear at the start that I am attempting to appraise the relative importance of health education in the public health field as compared with other elements. I am not attempting an analysis from a psychologic or other angle, of what constitutes effective health education or what technics will or will not arouse real interest and action. This is another story, though indeed a very important one.

We might, in a few words, remind ourselves of the origin in time and in its interrelationships of the health educational aspect of our public health program. Recently Dr. W. W. Bauer has said that "All public health workers are health educators whether they know it or not." This is more nearly true today than at any time in the past, and it is at least a laudable aspiration. Still, there are even now some essential workers in the public health field who have little or nothing to do with health education, such as the laboratory man typing sputums in pneumococcic infections or examining blood specimens in sulfonamide therapy. What these men are doing is basic in the public health program but really cannot be called health education. The same would apply to the engineer operating a water purification or sewage treatment plant, and in the main to the research work that developed the antigen for the pneumococcus vaccine or that originally purified the antibody in serum therapy. These men may do extremely important work and still not undertake even professional teaching activities, let alone popular health education.

As a matter of fact, health education played little or no part in the beginning eras of modern public health. It came into play, for obvious reasons, as public health evolved into the period of preventive medicine, where the attainment of disease preventive objectives (particularly in a democracy) is dependent on individual motivation. But back in the sanitary era the typhoid epidemics and other sanitary catastrophes of that earlier time were in fact the "educators" of the citizens and of the voters, inducing them, among other things, to appropriate adequate funds for the collective provision of sanitary measures, such as those that insure a safe water supply. But the community collectively did the job for the individual.

On the other hand, beyond the safe use of water to restrict the transmission of disease, the community cannot, by law, compel the individual to the proper constructive use of water for the attainment of certain hygienic and physiologic advantages. It cannot make him take enough water daily to meet the chemical needs of the body (though the importance of this has been somewhat overemphasized by health education); it cannot compel him to bathe with sufficient frequency, although here too health education has exaggerated the importance of the measure; it cannot even compel him to wash his hands with hot water and soap before he eats and after toilet. These uses of water must depend on personal information, on individual motivation—in fact, on health education (or, perhaps, on custom built on the commercial advertising of sanitary equipment, soap, and so on), though it must indeed be recognized that individual initiative won't be effective unless the water and the necessary utensils also are available.

In any event, we are now in the age of personal hygiene and preventive medicine, where health education comes fully into its own. Yet this modern phase of public health grew out of and is based on sanitation and is, in fact, the superstructure of public health. Health education is an essential part in that superstructure. How is it built into that framework? How much of a part does it play in the functioning of this whole public health mechanism? Let us ask ourselves these and a few other concrete questions.

WHAT CAN REASONABLY BE CLAIMED FOR HEALTH EDUCATION?

Well, there are some perfectly reasonable claims as to its accomplishments, though these claims are often confused, and the evidence is almost always fairly intangible. Yet we are all convinced that health education not only has important direct products to its credit but some by-products of outstanding importance.

By way of illustration, let us look for a moment at the diphtheria prevention program. Certainly health education of the lay public has contributed materially to the mortality improvement for this disease. Throughout the whole of 1940 Toronto, Ont., and Newark, N. J., two large cities, went without a single case of this disease. New York State, including the city and upstate area, with over 13,000,000 population, had several consecutive winter months in 1941 without a death from diphtheria. The million and more weekly industrial policyholders of the Metropolitan Life Insurance Company in the Pacific Coast and Mountain states went through the first eight months of this year without a diphtheria death. Certainly all of the literature, films, exhibits and lectures have played a big part in these accomplishments. Personal initiative by physicians, parents, teachers and others has been influential. Yet many

other elements have entered into the picture, such as the daily work of the private medical practitioner, the doctor's increasing use of prophylactic measures in his practice, the establishment of clinics, the production of biologic products and their distribution by communities, the postgraduate instruction of physicians with reference to antitoxin therapy as well as immunization and the like. While in fact the whole "struggle" has been in general educative, yet it is difficult in a situation of this kind to say just how much lay health education specifically has done. It can however be incidentally pointed out that, while it has been endeavoring to educate the public, it has also had a material effect on the medical mind as well. Many physicians first heard of the Schick test through lay contacts or through the perusal of literature primarily intended for lay consumption. This might be noted as a by-product of lay education.

One might take another example—the Framingham Tuberculosis Demonstration, where the tuberculosis death rate has declined from 121 per hundred thousand prior to the demonstration to 12.5 in 1940. Never before in the history of public health was a population so intimately and thoroughly exposed to health education as were the people of Framingham from 1916 onward. All conceivable educational channels were used and all important points in tuberculosis control stressed. The private medical practitioner participated wholeheartedly, and the public responded with a high degree of cooperation and understanding. Health education was an important factor in this program. Yet many other factors also were involved, even though health education may have had, directly or indirectly, something to do in securing and enhancing the effectiveness of each of them. I have in mind not only adequate appropriations but such essentials as the employment of full time medical staffs in schools and industries, the provision of adequate bed capacity for active cases, the proper direction of first class medical staffs in good sanatoriums, the establishment of local x-ray diagnostic equipment and milk pasteurization. There were obviously many basic items of sanitation and community equipment playing a part—brought about to some extent through health education and made more effective by health education but distinctly not in themselves health education.

IS IT EVER POSSIBLE ACCURATELY TO MEASURE HEALTH EDUCATION?

The agency with which I am associated, namely the Metropolitan Life Insurance Company, has carried on for years an extensive health educational program among its industrial policyholders. In this program it has used nearly all available devices, has had a fairly adequate budget and has enjoyed the cooperation of an exceptional instrument of education, namely the company's agency force in its contacts regularly with millions of homes. This work has now been going on for about thirty-two years. The results are to some extent measurable and are also quite favorable. It would seem that this health education program has borne fruit.

If the life expectancy at birth of these industrial policyholders is compared with that of the general population, it appears that since 1911 the policyholder group has gained about seventeen years in life expectation, as compared with about ten years for the country's population in general. It has also been calculated that the figure for lives saved in 1939 alone is approximately 112,000 in excess of the lives that would have been saved if the industrial policyholders had experienced only the mortality improvement since 1911 that has characterized the general population of the country. When this experience is broken down by causes of death, and when attention is paid particularly to those diseases at which health education has been especially aimed, we find, for instance, that since 1911 there has been a 92 per cent improvement in diphtheria mortality in the general population of the United States and a 95 per cent improvement in this industrial policyholder group—an excess policyholder improvement of 3 per cent. Without giving all the details, I may say that similar comparisons show an excess improvement in pneumonia of 7 per cent, in tuberculosis of 10 per cent, in diseases of the puerperal state of 17 per cent, and in total accidents of 24 per cent.

Of course, this insured group has shared with the general population all of the other influences that have been prevalent in the country in addition to the company's own health educational program. However, there is a complicating factor, and that is the special nursing service provided by the company for acutely ill policyholders and aimed particularly at problems of the industrial insurance group. In this nursing service special attention has been paid to diseases of the puerperal state and communicable diseases, including diphtheria and pneumonia. How much this nursing service, instrumental as it has been in also encouraging adequate medical care, may have influenced this mortality experience, it is difficult to say. Equally difficult is it, therefore, to appraise separately the health educational values.

There is one element in the Metropolitan experience that may be a more direct and more sensitive measure of health education than any of the disease factors cited, and that is the experience with appendicitis. Beginning in 1934 the company initiated an educational program aimed at the prevention of unnecessary fatalities from this disease. This has been carried out by means of literature, advertisements, posters, lectures and news releases. The cooperation of the medical profession, and particularly of the company's agents and medical examiners, has been forthcoming. During this period the company has experienced among its industrial policyholders an improvement in appendicitis mortality of over 30 per cent, and a drop of 40 per cent since 1929. The rate has been declining rapidly and is still declining. (Incidentally, the general population improvement in this period since 1934 has been about 24 per cent.) Other reports, from hospital studies in Philadelphia, for instance, show a decline in perforated appendicitis from 43 per cent in 1928-1929 to 19 per cent in 1937. Moreover, into this picture have come no great new medical discoveries, such as toxoid in diphtheria, serum and chemotherapy in pneumonia, or immunization procedures as in other communicable diseases, although there may have been some modest advances in surgical skill, some stimulus to hospitalization through insurance plans, and, very recently, some experimental use with sulfonamide derivatives and other products in the treatment of peritonitis following perforation. No important changes have taken place in diagnostic or therapeutic practices. One might say that practically the only outstanding new factor in the picture, in relation to the other constants or changing minor variables, has been this intensive educational program. Perhaps, therefore, this might be offered as a fairly good example of a health educational accom-

plishment. Practically all other examples that one can think of share the limitations pointed out not long ago by Dr. Martha C. Hardy, who stated that "Evaluation technics in health education . . . are very limited. . . . These results, and reports from other investigations, while interesting, seldom permit conclusive answers. They are much more likely to raise questions than they are to produce evidence of the effects of health education."

ARE HEALTH EDUCATORS SOMETIMES CONFUSED AS TO DEFINITION?

I need perhaps quote only one or two examples to indicate positive answers to this question. One who has contributed a great deal indeed to physical education recently said that "Health education in schools has three major lines of effort: health service, healthful living in the school, and health instruction." I expect most readers will agree that this does not seem to be a very accurate or clear concept. This leader then goes on to explain what he means by each of these divisions of effort. He says, for instance, that under "health service" he means "examination and protection of children." Under this heading he includes also the search for defects and structural disease and the examination of "attitudes, personality traits and responses, as well as defects and organic health disturbances." In other words, he lumps under one of these phases of "health education" all of the professional, technical, medical and clinical procedures in the schools. Obviously these procedures are not health education, though of course they should be health educational.

IS THE SIGNIFICANCE OF HEALTH EDUCATION SOMETIMES EXAGGERATED AND ITS SCOPE MAGNIFIED OUT OF PROPORTION TO REALITY?

The attitude reflected in the foregoing quotations would seem to be one example of a clearly affirmative answer to this question. Others will probably occur to most of us. If I may carelessly employ metaphors, many have been impressed with the tendency on the part of health educators to "put the cart before the horse" or at any rate to confuse the part for the whole. This may be in part due to two factors:

1. Professionally trained people have often approached health education through highly specialized channels, such as those of physical education. They consequently seem to acquire an out of focus view or a tangential rather than an inherent relationship to the public health fields.

2. For nonprofessionally trained workers, health education has been the back door into the barn of preventive medicine, as easy access for those with no specific technical training for any of the basic jobs in public health and preventive medicine. In spite of this, or perhaps because of it, those entering by this door seem disposed to think that the barn and the door are one and the same thing.

An illustration of this confusion and of this identification of the part with the whole seemed to be implied in a recent request from a health educator that a paper be prepared on the subject "Do the Recent Draft Figures Show Serious Gaps in Health Education?" This arresting suggestion involved, apparently, two misconceptions: (1) that health education can ordinarily be measured by its effects and (2) that health education was the important if not the only factor (or should have been) in determining the results of the current draft figures, as compared with those of twenty-five years ago.

Of course, the draft figures will be turned in all directions. Those who want to use them for the further extension of socialized services are already loudly lamenting the allegedly poor state of the nation's health (which really was never better) and will no doubt find ammunition that can be diverted to this end. Those who wish to "view with alarm" will point out that more are being rejected today than in 1917, but this comparison is unwarranted, because today we started with the idea of careful selection for training, using higher standards and more delicate methods. More are being rejected for certain causes, such as ear and dental defects. From this it may be claimed that our whole health effort and our machinery for medical care have been ineffective and that health education has completely failed, yet we know this to be a gross misinterpretation. On the other hand, those disposed to take a favorable view will find plenty of supporting evidence. In fact, the tuberculosis figures have already been pointed to as evidence of the value of health education in this intervening period, in view of the rejection in 1917 of higher percentages for pulmonary ailments than in 1941. Very probably health education has played a part in this situation, but how important also have been a great variety of other factors, such as isolation and hospitalization, improvements in x-ray technic and fluoroscopy, pneumothorax therapy, milk pasteurization, advances in nutrition, and higher economic living standards?

Confusion of definition, magnification of importance and misunderstanding as to measurability sometimes seem to lead to an attitude that assumes not only that all health activities are a part of health education but also to the belief that only health educational activities should be tolerated in schools and elsewhere. For instance, one finds in an editorial in a recent number of the *Journal of School Health* a statement that "All health services practiced in schools can and should be educational." No one would quarrel with that. The editorial goes on, however, to imply its acceptances of the corollary that "if the service cannot be made educational it should not be in the schools at all." This is to ignore certain basic essentials in school health programs. We should not forget that the medical examinations of children in schools is done in part to apply technical skills with the object of discovering early disease, of finding defects that can be corrected, of controlling the spread of communicable diseases and of disclosing personal practices that have a bearing on the physical and mental status of the child. The basic service here is a clinical, epidemiologic and physiologic one. It has some value, whether educational or not. Of course, these procedures should always be used for constructive health educational purposes, but that is not their sole or primary purpose though it may become their greatest measure of utility in highly sanitated communities.

HAS HEALTH EDUCATION SOMETIMES BEEN MISLEADING AND OBSTRUCTIVE?

It seems likely that there are examples warranting an affirmative answer to this question. Many will remember the great emphasis of two decades ago on toothbrushing as a means for the prevention of dental decay. Not only was the child taught in school cer-

tain highly detailed technics as regards toothbrushing, but whole schools and squads of children were mobilized in playgrounds and elsewhere and exposed to toothbrush drills, thus overemphasizing this already exaggerated point. The same amount of energy, time and funds diffused nationally through this program, if concentrated into a limited field of research, let us say into the relationship of nutrition or of heredity to dental caries, might have produced constructive results, and at any rate probably would not have been largely wasted.

HAS HEALTH EDUCATION SOMETIMES FAILED?

If health education has failures in its record, it is probably because of misapplication, such as in the aforementioned toothbrush drill era, or it is because, through its own megalomania, it "bites off more than it can chew." An example in the latter category may well be the experience in the matter of disease control in the province of Quebec. In this province death rates from controllable disease, such as pneumonia, tuberculosis and diphtheria, still remain much higher than in the other Canadian provinces or in the United States. These diseases refuse to respond to educational attack. Such control efforts under identical epidemic conditions are much less successful than elsewhere.

By way of illustration, let us take the recent influenzal pneumonia outbreak, which early in 1941 spread across both Canada and the United States, from west to east. In most parts of the two countries, in spite of the increase in influenza with accompanying increase in the incidence of pneumonia, the pneumonia death rate for this period in 1941, as compared with 1940, did not increase but actually decreased, evidently reflecting the effectiveness of serum therapy and chemotherapy. In contrast, in Quebec province there was not only an increase in influenza and pneumonia incidence, but a sharp increase in pneumonia fatalities—quite obviously for the reason that medical practice and public health procedures in this province are backward and that the province does not yet have, in comparison with other areas, these therapeutic advantages in meeting the pneumonia problem.

Now, what will bring pneumonia under control in Quebec province? Health education? What will make modern diagnostic and therapeutic procedures available? Health education? It may help a little in all directions but it will not solve the problem. As a matter of fact, a great deal of health education is being done and has been done in this province. The Metropolitan Life Insurance Company has concentrated on these questions there even to the extent of preparing health education material in the native language. It insures a larger percentage of the population there than elsewhere. It has a higher industrial policyholder mortality rate there than elsewhere. It and other agencies have tried health education, but that does not meet the need.

What is needed is a whole set of basic facilities and services, such as better equipped and staffed medical schools, improved medical curriculums, better undergraduate and postgraduate medical education; better basic training for nurses, more competent public health training for nurses; nonpolitical public health administration; more adequate appropriations, more adequate supplies of biologic products for use free or at cost; a greater awareness on the part of physicians of the preventive medical point of view; more adequate hospital facilities; better and more numerous physical plants for clinical services, and so on. While health education has a bearing on all of these things and will help to make them available and to get them used when available, it will not in itself produce them or solve the problem without them. Under present conditions in this province its efforts are largely wasted and doomed to partial failure.

HOW SERIOUSLY SHOULD HEALTH EDUCATORS TAKE THEMSELVES?

From the foregoing the implication would seem to be that we should not take ourselves too seriously. We should remember that health education is one of a number of things. Health educators are probably feeling an unwarranted anxiety in these trying times. They are for instance, watching the draft figures anxiously. They have "staked everything" on producing results through education, and the present generation, coming up for examination, is really their first child to reach maturity. Nobody knows, because we have never had any adequate method of measurement, just how well he has learned his lessons. Health educators, according to their disposition, are now anticipating feeling very good or very bad when the figures begin, in final form, to come through. Yet they should remember that they are dealing with one factor only, with one of the most intangible of elements and the hardest one to measure. Should conditions prove to be better or worse, there would be nothing to crow or to weep about from the angle of health education or from any other single angle in the picture, as too many elements share this responsibility.

WHAT THEN IS THE CHARACTER OF HEALTH EDUCATION AND ITS RELATION TO PUBLIC HEALTH?

While the element health education is not all important, it is important to all other elements in public health and preventive medicine as practiced in the modern community. Health education helps to create its associate units essential in a basic health program. It should permeate all of these related services and influences. As a matter of fact, without these concrete services, such as medical examinations in schools, clinic facilities in health centers and examination services in industry, health education remains a highly intangible, theoretical, abstract and largely futile commodity. It finds its substantiation, its realization in its relationship to these practical services. It is visualized, precipitated, brought out of the realm of theory by its contact with its basic service colleagues. It therefore has a creatively reciprocal relationship with the other elements in public health. It enhances their value and reality and finds its own realization through them. It is the one element that contributes most to making the whole substantially greater than the sum of its parts.

HAS HEALTH EDUCATION A UNIQUE SIGNIFICANCE?

While health education is one of many elements in the public health picture, it is different in character from any of these elements. It is not just "one other thing" in preventive medicine. It is or should be an ingredient in all the basic services in preventive medicine. All other services should be shot through with it or illuminated by it. It is actually or potentially inherent in all of them. It might perhaps be called the

enzyme in the process. In the digestive process are involved the mucous membrane of the gastrointestinal tract, the cells of the villi of the intestinal walls, the blood vessels, the cells of the blood stream, the musculature and the constituents of the food itself. These all have to be there and all have to do their part. They constitute the basic essentials. But their efficiency and thoroughness are stimulated and released by the effect of the digestive enzyme on them. Now in public health, within the school walls for instance, health education bears a similar relationship to the doctor, the nurse, the clinic, the laboratory and the other personnel and equipment. Perhaps the analogy has flaws—doctors are apt to do some good in just examining and treating children, with little or no health educational motive, while digestion won't take place at all without the enzyme. Nevertheless in schools, in health centers and clinics, in industry and in all phases of the public health program it is the enzyme "health education" that implements and activates its associate forces in the cooperative effort to prevent disease, promote health and build the vigor and endurance demanded by the stress of living in our time.

All that I have said applies to health education in a democracy—in a free society, where initiative, judgment and individual decision and enterprise still function—where it is still possible and indeed necessary to try to inform and persuade people as to what they should do for the benefit of themselves and the community. On the other hand, health education and indeed all education as we know it are of disappearing importance in most of the rest of the world, where the "educator," turned propagandist, tells the people what to do, although even in Germany the chief propagandist is said recently to have lamented the fact that there were still a few people in his country who could not be told how to act and live and think.

Health education, and indeed all education as we think of it in America, is not only an essential ingredient in the free democratic process. Today, more than ever before, it must play a vital part in sustaining and justifying our way of living. As Hutchins has said, "What the world needs, what this country must have, is free minds . . . minds informed by principles derived through human experience through the ages, minds that are open no matter what waves of change beat upon them." Such education, in all fields, including our own, is in the long run our chief defense against the growing trends toward socialization, collectivism, dictatorship and totalitarianism, both abroad and at home.

1 Madison Avenue.

The President's Birthday Ball.—The support of research through public subscription such as the President's birthday ball or the Christmas seal campaign of the National Tuberculosis Association presents special features worth comment. It would be a splendid tradition if each president could designate on what disease the proceeds of the homage tendered him should be spent. Then with an occasional change of presidents we should see a reasonable distribution of support to different forms of research. But the limitation of this form of support is that the machinery of securing the funds may become effective more rapidly than the organization to expend them, and the desire for specific results may exceed the probable returns. A better procedure than usually characterizes public subscriptions would be for each of the specialist societies to nominate research councils entrusted with the expenditure of funds collected by public subscription.—Gregg, Alan: The Furtherance of Medical Research, New Haven, Conn., Yale University Press, 1941.

AIR EMBOLISM IN ARTIFICIAL PNEUMOTHORAX

THOMAS R. JONES, M.D.
HOUSTON, TEXAS
AND
JESSIE A. LOCKHART, M.D.
SANATORIUM, TEXAS

Of all the complications of artificial pneumothorax therapy, air embolism is the most alarming and most dangerous. Its occurrence is sudden and without preceding signs or symptoms, as it is in reality an accident. Its results either are instantaneous or appear in a short time.

Rogers, while irrigating the empyema cavity of a small girl in 1864, observed an attack characterized by pallor, unconsciousness and clonic convulsions with recovery after twenty-four hours. While diagnosed as pleural eclampsia it may be logically assumed from the description of the attack that this was a case of cerebral air embolism.

Brandes[1] in 1912, while attempting to outline an empyema cavity with bismuth paste, accidentally demonstrated the mechanism of air embolism. On injection of the paste, the patient became unconscious and was suddenly seized with fatal convulsions. On postmortem examination bismuth was found in the vessels of the cerebral cortex and brain stem.

AIR EMBOLISM IN PERIPHERAL CIRCULATION

Every physician and intern who has given intravenous injections knows that often small quantities of air have been introduced into the peripheral veins with no untoward effect.[2] This can be explained on the basis that the amount of air injected was small and its injection into the circulation slow. This allowed for its absorption in the vascular lung tissue. Thus, 3,910 cc. of air was injected slowly into a dog over a period of eighty-seven hours before death occurred.[3]

On the other hand, fatal air embolism does occur during operations on the neck and in manual loosening of placentas. Here the quantity of air admitted is large and its entrance into the circulation rapid. Experimental work has shown that the fatal dose of air for a 150 pound (68 Kg.) man would be about 525 cc., if injected rapidly into the peripheral circulation.[4] Death is the result of failure of the right side of the heart due to blocking of the pulmonary circulation and interference with ventricular output of the heart due to air bubbles.[5]

PHYSIOLOGY

Curtillet,[6] in experimental work on animals, has shown that air bubbles do not reach vessels of a caliber of less than 30 microns; that is to say, they never reach capillaries. Arrested in the endarterioles of 30 to 40 microns in diameter, they are rapidly absorbed. Air and oxygen are absorbed at about equal rates of speed. Carbon dioxide is absorbed more rapidly than either. Air bubbles injected into the peripheral circulation, barring such anatomic defects as a patent foramen

From the Texas Tuberculosis Sanatorium, Sanatorium, Tom Green County, Texas.

1. Brandes: München. med. Wchnschr. **59**: 2392, 1912.
2. Reyer, G. W., and Kohl, H. W.: Air Embolism Complicating Thoracic Surgery, J. A. M. A. **87**: 1626 (Nov.) 1926.
3. Richardson, H. F.; Coles, B. C., and Hall, D. E.: Experimental Gas Embolism, Canad. M. A. J. **30**: 584 (June) 1937.
4. Moore, R. M., and Braselton, C. W.: Experiments in Gas Embolism, Bull. John Sealy Hosp. & Sch. of Med. **1**: 78 (May) 1939.
5. McDaniel, Shaw: Air Embolism: Its Cause and Treatment, Dis. of Chest **7**: 146 (April) 1941.
6. Curtillet, E.: Arterial Air Embolism, J. de chir. **53**: 461 (April) 1939.

ovale or a ductus arteriosus, are completely filtered out in the vascular system of the lungs and are never found in the vulnerable arterial circulation.

AIR EMBOLISM IN ARTERIAL CIRCULATION

It has been demonstrated that the injection of moderate amounts of air into a systemic vein leads to embolism of the smaller vessels of the lung with no harmful defects. On the other hand, relatively minute quantities of air injected into the pulmonary vein produce severe reactions or death. This is because the air passes from the vein, through the left side of the heart into the systemic arteries. Experimental work [7] indicates that the lethal dose of air thus entering the arterial circulation of a 150 pound man is about 37.5 cc. This depends on how rapidly air emboli obstruct vital cerebral or coronary arteries.

From clinical observations it seems that cerebral embolism is the most frequent cause of death in these cases. However, Moore and Braselton, working with cats, found that death occurred from coronary obstruction in every case, regardless of the position of the animal. Within a second or two after injection of air into the animals, air bubbles were seen descending the coronary arteries. The air filled both the left and the right arterial tree. This complete obstruction of the arteries led to extreme dilatation of the ventricles in five to six minutes, though under artificial respiration the animals made convulsive respiratory movements at this time indicating that the respiratory center was still functioning.

Wever [8] demonstrated by numerous experiments on rabbits, dogs and cats the effects of introducing air directly into the carotid arteries and found that the symptom complex resulting coincides with that sometimes observed in air embolism from pneumothorax therapy. At times he could produce air emboli with less than 1 cc. of air, while on other occasions the injection of 2.3 cc. of air failed to produce symptoms. Depending on the amount of air introduced, he observed variations from mild tremors and mild disturbances of cardiac and respiratory action to sudden death. The milder effects resembled what we believe has been incorrectly termed pleural shock.

AIR EMBOLISM VERSUS PLEURAL SHOCK

Many physicians [9] still diagnose as pleural shock cases in which the syndrome of pallor, nausea, vertigo, convulsive tremors, coma and temporary blindness is present, with complete recovery.

Schlaepfer [10] carried out a series of experiments on the normal pleurae of various animals. He found that stimulation of a normal pleura by mechanical, chemical, electrical and other stimuli did not produce a constant specific reaction by way of the central nervous system as one would expect if the so-called pleural reflexes were the source of the syndrome mentioned.

Capps [9] repeated these experiments on dogs with an acute pleurisy produced by intrapleural injections of turpentine and oil contaminated with bacteria. In some of these cases, stimulation of the inflamed visceral pleurae produced no decided change in blood pressure. In others there was a considerable fall of blood pressure, which was sometimes fatal. Capps also stated that during aspirations of pleural effusions in human beings he had purposely irritated the visceral pleura with the end of a trocar and had observed a significant fall of blood pressure. During several hundred aspirations of simple pleural effusions and tuberculous empyemas at the Texas Sanatorium, we have frequently either punctured or scratched the moving visceral pleura with the trocar. In none of these cases have we ever observed syncope or a significant fall of blood pressure from irritation of the visceral pleura.

Introduction of the trocar through the parietal pleura without anesthesia produces excruciating pain; however, after the trocar has pierced and anesthetized the parietal pleura, it may pierce or scratch the visceral pleura without producing any perceptible sensation of pain or discomfort. Shaw [11] states that during several hundred intrapleural pneumolyses and thoracoscopies of the chest, both with and without manipulation of the visceral pleura, he has never observed a case of pleural shock or air embolism. During these intrapleural pneumolyses adhesions are severed by the actual cautery, at times while work is being done close to both the parietal and visceral pleurae. If such a mechanism of pleural reflexes through the central nervous system existed, the mechanical and thermal stimulation of intrapleural pneumolyses should certainly produce pleural shock. Apparently the absence of reactions in these cases is due to the fact that the visceral pleura and lung are never actually pierced and there is no chance for cerebral air embolism to occur.

PATHOLOGY

It is often impossible to obtain pathologic-anatomic proof in fatal cases of air embolism because of the fact that minimal and undemonstrable air bubbles introduced in the carotid arteries often cause death.[12] Frequently, however, at autopsy air bubbles have been found in the cerebral vessels of fatal air embolism cases.

Spielmeyer [13] has reported that the first demonstrable pathologic changes in the brain appear about fifteen hours after the air is injected and consist of a beginning liquefaction of brain cells and the encrustation of the fine nets around the ganglion cells. After three to five days, proliferation of glia cells at the site of the diseased nerve cells is very pronounced. These changes are predominantly in the form of islands and there are no bleedings or necrotic softenings. About twelve days after the air embolism occurs, large glia cells are seen at the place of the disintegrated cortex cells.

Air embolism is more apt to occur in the pathologically altered lung than in the normal lung because, in the presence of induration and adhesions in the former, vascular contractility is impaired. In the infiltrated portion of the tuberculous lung the pulmonary veins are kept stretched in the rigid tissues and cannot evade the needle as in the healthy tissues. Should the needle injure such a vein, its walls cannot collapse as they would normally.[6] It is evident that, when to such

7. Moore, R. M., and Braselton, C. W.: Ann. Surg. **112**: 212 (Aug.) 1940.

8. Wever: Cerebrale Luftembolie, Beitr. z. Klin. d. Tuberk. **31**: 159, 1914.

9. Capps, J. A.: Air Embolism versus Pleural Reflex as the Cause of Pleural Shock, J. A. M. A. **109**: 852 (Sept. 11) 1937. Nepi, A. D.: Pleural Eclampsia During Artificial Pneumothorax, Am. Rev. Tuberc. **43**: 295 (Feb.) 1941. Cocke, C. H.: Pleural Shock, ibid. **24**: 545 (Nov.) 1931.

10. Schlaepfer, K.: Air Embolism Following Various Diagnostic or Therapeutic Procedures in Diseases of the Pleura and Lung, Bull. John Hopkins Hosp. **33**: 321 (Oct.) 1922.

11. Shaw, Robert: Personal communication to the authors.

12. Kowitz: Luftembolie oder Pleurashock, Beitr. z. Klin. d. Tuberk. **50**: 374, 1922.

13. Spielmeyer, quoted by Pollak, Maxim: Air Embolus, Am. Rev. Tuberc. **28**: 187 (Aug.) 1933.

an injury a negative pulmonary venous pressure of —7 to —8 cm. of water is added, air may be sucked into such an injured vein from the needle tubing, from the lung alveoli or from the pleural cavity itself. It is not necessary that air should be introduced into the vessel from the outside to produce air embolism.

INCIDENCE

Among 1,869 cases of pneumothorax reported in the literature [14] there occurred 43 cases of air embolism. This is an incidence of 2.3 per cent, or 1 in every 43 cases of pneumothorax. Among 126,547 pneumothorax treatments reported [15] there occurred 96 cases of air embolism. This is an incidence of 0.0758 per cent, or 1 during every 1,318 pneumothorax treatments. Among 142 cases in which the mortality was reported [16] there were 41 deaths. This is a mortality of 28.8 per cent for all cases of air embolism.

At the Texas Sanatorium during the past twenty-three years 115,380 pneumothorax treatments have been given with 19 cases of air embolism. This is an incidence of 0.016 per cent, or 1 air embolism in every 6,073 pneumothorax treatments. During this period in which 19 air embolisms occurred there were 3,036 pneumothorax patients receiving treatments. This is an incidence of 0.62 per cent, or 1 air embolism in every 159 pneumothorax patients. Among these 19 cases there were 7 deaths, a mortality of 36.8 per cent.

REPORT OF CASES

Among the 19 cases of air embolism occurring at the Texas Sanatorium, a fairly complete record was available in 17 cases:

CASE 1.—R. H., a white man aged 25, a clerk, with no history of exposure to tuberculosis, had influenza in December 1940 with poor recovery, followed by malaise, pleurisy and loss of weight. Gradually he acquired a productive cough and by February was raising 4 cc. of mucopurulent sputum daily. Fever and intermittent hoarseness appeared. On Feb. 20, 1941 he stopped work and began observing regular rest hours. On admission to the sanatorium ten days later, fairly extensive involvement of the upper lobe of the left lung was found with no definite cavitation. He was classified moderately advanced (b), and the sputum contained tubercle bacilli. Left artificial pneumothorax was advised, and the initial puncture of 300 cc. of air was given on March 14 with good manometer readings. Next day 400 cc. of air was given with a final manometer reading of 0, —8 (cm. of water). The third refill was given March 18 at 8:40 a. m. As the pneumothorax needle entered the chest the manometer showed a pressure of plus 6 cm. of water. The needle was manipulated and the manometer showed a reading of —12 cm. A few cubic centimeters of air were given and the reading was —8, a few more cubic centimeters and the reading was —6, a few more cubic centimeters and the reading was —2. The patient became pale, closed his eyes and rolled forward slightly. He became unconscious, the pupils dilated, and the eyes rolled upward. He made three or four feeble, jerking movements of the left arm and then lay still. Epinephrine hydrochloride and artificial respiration were administered. Irregular pulse beats appeared, and these were followed by several convulsive respiratory movements. At 8:55 a. m. both the heart action and the respiratory movements stopped. In all about 50 cc. of air was induced, and the fatal reaction lasted twelve minutes.

At necropsy the left lung was almost completely collapsed by pneumothorax. A single sheet adhesion extended from the apex to the first rib. Three needle punctures with subpleural hemorrhage were seen in the arteriolateral aspect of the lower lobe. Section of the lung showed that one needle tract passed through a 5 mm. pulmonary vein. No air bubbles could be demonstrated in the coronary or cerebral arteries. The heart was contracted and there was about 1 ounce (30 cc.) of blood tinged fluid in the pericardial sac.

CASE 2.—C. H., a white woman aged 19, a student, with no history of exposure to tuberculosis, suffered from malaise and loss of weight in November 1938, and a slight cough appeared. One year later a diagnosis of tuberculosis was made. On admission to the sanatorium, Nov. 21, 1939, her condition was classified as far advanced (a), with extensive involvement of the upper half of both lungs with a small cavity in the left upper lobe. The sputum contained tubercle bacilli.

The right lung improved steadily, and on July 31, 1940 an unsuccessful attempt was made to collapse the left lung by artificial pneumothorax. On a second attempt two days later, 350 cc. of air was introduced with a final manometer reading of 0, plus 2 cm. of water. On the third refill, March 5, the initial reading was —1, —7 cm. of water; 25 cc. of air was given and the manometer read 0, plus 7 cm. of water. The needle was withdrawn, and the patient rose to a sitting position. She complained of feeling weak and nauseated and immediately had a convulsive seizure of the right arm and leg and became unconscious. Both eyes were deviated to the left side. The head was lowered, and artificial respiration was given. She regained consciousness in about ten minutes but complained of blurred vision and severe headaches for the next twelve hours.

CASE 3.—H. G., a white woman aged 23, a housewife, with no known exposure to tuberculosis, had pleurisy in 1937, and after the birth of a child in September 1939 she had malaise and loss of weight. In May 1940 she became ill with a chill and high fever, and this was followed by a productive cough and expectoration of about 30 cc. of mucopurulent sputum daily. Hoarseness, night sweats and indigestion appeared. She remained in bed until her admission to the sanatorium, June 12, 1940. Her condition was classified far advanced (b), with extensive disease, and multiple cavitations in the left lung. The sputum was strongly positive for tubercle bacilli. Left artificial pneumothorax was advised, and the initial puncture was made on June 17 at 9:20 a. m. On the first puncture the pleural space was not located and there was no oscillation of the manometer. A second puncture was done immediately, and the manometer showed a negative pressure of —4 cm. of water. The air was turned on and off twice. The pressure was still —4, but there was no fluctuation of the manometer on respiration. In all, about 25 cc. of air had been injected when the patient closed her eyes, became pale and slumped forward. She became unconscious, and feeble tonic tremors occurred momentarily in the arms and legs. Epinephrine hydrochloride and artificial respiration were given and the head was lowered. The patient improved for a minute and was able to talk. She then became unconscious again. The respiration was gasping and convulsive and the pulse was irregular. The condition rapidly grew worse and the heart action ceased at 9:46 a. m. despite artificial respiration. There was no necropsy.

CASE 4.—E. T., a white man aged 27, a café manager, had been exposed to a tuberculous father. In March 1939 malaise

14. Forlanini: Gazz. d. osp., November 1882. Myers, J. A.; Levine, Ida, and Leggett, Elizabeth A.: Air Embolism and Spontaneous Pneumothorax Complicating Artificial Pneumothorax, Brit. J. Tuberc. **31:** 77 (April) 1937. Andrews, C. H.: Cerebral Air Embolism Complicating Artificial Pneumothorax, Am. Rev. Tuberc. **23:** 435 (April) 1931. Sacks, quoted by Cocke.[9]

15. Bruns, E. H.: Air Embolism As a Complication in Artificial Pneumothorax Therapy, Colorado Med. **27:** 237 (May) 1930. Tice, Frederick, and Hruby, A. J.: Collapse Therapy at Chicago Municipal Tuberculosis Sanitarium, J. A. M. A. **113:** 101 (July) 1939. Sangman: Beitr. z. Klin. d. Tuberk. **31:** 571, 1914. Motz, P. B.: Am. J. M. Sc. **176:** 87 (July) 1928. Stivelman, B P.: Am. J. M. Sc. **145:** 6, 1923. Cocke.[9] Forlanini.[14] Andrews.[14]

16. Warring, F. C., and Thomas, R. M.: Spontaneous Air Embolism, Am. Rev. Tuberc. **42:** 682 (Nov.) 1940. McCurdy, Thomas: Air Embolism Complicating Artificial Pneumothorax, ibid. **30:** 88 (July) 1934. Bishop, H. A.: Air Embolism and Artificial Pneumothorax, ibid. **10:** 591 (Jan.) 1925. Schattenberg, H. J., and Ziskind, Joseph: Air Embolus as Complication of Artificial Pneumothorax, Am. J. Clin. Path. **9:** 477 (July) 1939. Walsh, Frank B., and Goldberg, H. K.: Blindness Due to Air Embolism: A Complication of Extrapleural Pneumolysis, J. A. M. A. **114:** 654 (Feb.) 1940. Hall, W. E. B.: Left Heart Arterial Air Embolism, ibid. **109:** 125 (July) 1937. Reyer and Kohl.[2] Spielmeyer.[2] Myers, Levine and Leggett.[14] Andrews.[14] Bruns.[15] Tice and Rhuby.[15]

and slight expectoration appeared. On entering the sanatorium on May 10, 1939 he was classified as having moderately advanced (c) tuberculosis, showing extensive involvement of the left lung with cavitation in the upper lobe of the left lung. The sputum contained tubercle bacilli. Left artificial pneumothorax was advised and 350 cc. of air was given at the initial puncture on May 17. The eighteenth refill of 300 cc. was given on July 21, with an initial reading of 0, —8, and a final reading of 0, —4 cm. of water pressure. The patient felt a moderate pain in the left chest but walked back to his room. Here he became nauseated, dizzy and developed blurred vision and headache. The blurred vision and headache lasted for fifteen hours, when complete recovery occurred.

CASE 5.—J. B. B., a white man aged 27, an oil field worker, with no known exposure to tuberculosis, suddenly had a massive hemorrhage on March 10, 1939. He was given right artificial pneumothorax and went back to work immediately. In August 1939 he began streaking again and this time was given pneumothorax on the left side. He observed no rest hours prior to his admission to the sanatorium on September 17. His condition was classified as moderately advanced (a); there was bilateral cavitation in both apexes. The sputum was positive for tubercle bacilli, and there was an anal fistula. Bilateral pneumothorax was continued, and on October 11 the pneumothorax needle was induced into the right chest. Before any air could be given the patient complained of blindness, nausea, chilly sensations, and pains in both arms and the right chest. The needle was withdrawn, and the head lowered. The patient became semiconscious, and tonic contractions of both arms appeared. Little blue cyanotic blotches about 1 cm. in diameter appeared over the right anterior chest and right arm and lasted for about fifteen minutes. Severe headache, nausea and blurred vision continued for twelve hours, followed by complete recovery.

CASE 6.—L. C. L., a white man aged 58, a salesman, with no known exposure to tuberculosis, acquired malaise and loss of weight in March 1937, and two months later a diagnosis of pulmonary tuberculosis was made. On admission to the sanatorium, Aug. 11, 1937, his condition was classified as far advanced (a), showing extensive involvement of the left lung with cavitation and an apical lesion of the right lung. The sputum contained tubercle bacilli. He failed to improve on bed rest, and left pneumothorax was advised. The initial injection of 400 cc. of air was given on September 15, with poor manometer readings. Two days later a second refill of 200 cc. of air was given. On September 20 the pneumothorax needle was induced for a third refill. As the needle entered the pleura there was a manometer reading of —3 cm. of water, but no air was injected. The patient complained of nausea and dizziness. He became unconscious, and clonic and tonic convulsions appeared. The head was lowered, and epinephrine hydrochloride was given. The patient regained consciousness for about one minute, but this was followed by another convulsion. He became cyanotic, and irregular respiration followed; the pulse was regular. Artificial respiration and caffeine with sodium benzoate were given, but the convulsions became worse and the patient died after a reaction of about thirty minutes. No necropsy was performed.

CASE 7.—Mrs. M. W., a white woman aged 25, a housekeeper, whose husband died of tuberculosis in 1925, had malaise, cough and expectoration in October 1935. She entered the sanatorium on Feb. 25, 1936 and her condition was classified as far advanced (b). There was a very extensive involvement of the left lung with an apical lesion of the right lung. The sputum was positive for tubercle bacilli. Left artificial pneumothorax was advised, and the initial injection of 250 cc. of air was given on March 4, 1936. Two days later an attempted refill was unsuccessful because of inability to find the pleural space. Later, on the third attempt to give a refill, the pleural space again could not be located, and after some manipulation the needle was withdrawn. The patient immediately complained of vertigo and nausea. She became unconscious and had generalized convulsions. The head was lowered, and epinephrine, caffeine and artificial respiration were given. The convulsions increased in severity and occurred more often. The heart beat regularly for sixty minutes and then became irregular and stopped in five more minutes. No necropsy was performed.

CASE 8.—A. L., a white man aged 42, a farmer, had malaise and bloody expectoration in 1922. On admission to the sanatorium, Sept. 3, 1924, his condition was classified as far advanced with total fibrocavernous involvement of the left lung and scattered small lesions over the right lung. The sputum contained tubercle bacilli. Left artificial pneumothorax was begun on November 24 with poor manometer oscillations. The fifth refill was given on December 8. After 50 cc. of air had been injected the patient became unconscious, and generalized convulsions followed. Morphine and epinephrine were given, but the convulsions became more severe, and death occurred in thirty minutes.

CASE 9.—M. W., a white woman, a housewife, had taken pneumothorax in the sanatorium for six months and had been discharged for ambulatory treatment. On a subsequent refill the pneumothorax needle was introduced and about 200 cc. of air had been given. Suddenly the patient became limp and rolled forward unconscious. A few quivering twitches passed through the body, but there was at no time any type of convulsion. The pulse was weak and irregular and soon became imperceptible. The heart ceased beating in about five minutes.

CASE 10.—J. L. D., a white woman aged 30, a housewife, had taken artificial pneumothorax therapy for slightly more than two months, when the needle was introduced for the tenth refill. The manometer showed unsatisfactory oscillations and after 100 cc. of air showed a positive pressure. The needle was withdrawn, and the patient arose to a sitting position. She immediately complained of vertigo and nausea and lay down on the operating table. She became unconscious and was seized by tonic convulsions and lost control of the urinary sphincter. The head was lowered and artificial respiration was applied. Consciousness returned in about ten minutes, but at this time she complained of severe headache and blurred vision. The right pupil was dilated for eight hours and the right side of the face showed the presence of a spastic paralysis for twenty-four hours. The headache remained for forty-eight hours, during which time the foot of the bed was kept elevated.

CASE 11.—T. B., a white woman aged 24, a nurse, had taken artificial pneumothorax therapy for about one year with poor manometer fluctuations, when the needle was induced for a refill. The operator was unable to locate the pleural space, and after some manipulation the needle was withdrawn. Immediately the needle was induced again through the next higher interspace. The manometer showed a negative pressure of —4 cm. of water, but the patient complained of nausea and faintness, and the right pupil became dilated. The patient became semiconscious for two or three minutes, and the pupil remained dilated for about ten minutes. A severe headache lasted for twelve hours, followed by complete recovery.

CASE 12.—Y. R., a woman aged 21, Mexican, a housewife, during the fifteenth refill on Nov. 18, 1940 complained of severe pain at the site of induction of the needle. The manometer showed a negative pressure of —6 cm. of water. After 25 cc. of air had been induced the manometer showed a positive pressure. The patient became limp, unconscious and cyanotic. The respiration was irregular and gasping for five minutes and then became regular. The patient became conscious but complained of feeling weak and faint for the next ten hours.

CASE 13.—M. B., a white woman aged 25, a housewife, had taken artificial pneumothorax therapy with good manometer oscillations for eight weeks. On the tenth refill, after about 50 cc. of air had been introduced, the patient suddenly was seized with severe tonic and clonic convulsions. Bloody, frothy mucus came from the mouth and nose. The patient bit her tongue several times and attempted to swallow it. Artificial respiration and epinephrine were given. The patient remained unconscious for two hours, during which time recurrent convulsions occurred and rectal and bladder sphincter control was

lost. At the end of this period the patient complained of blurred vision for the next twelve hours. Severe headaches persisted for the next three days.

CASE 14.—A white woman aged 23, a housewife, had received artificial pneumothorax therapy for eight weeks. On the eleventh refill the operator had trouble locating the pleural space but after some manipulation of the needle secured a manometer reading of —8 cm. of water. After 100 cc. or air had been given the patient became pale and unconscious and was seized with generalized convulsions. She uttered loud groaning sounds, vomited several times, and blood tinged, frothy saliva came from the mouth. The patient was given epinephrine and artificial respiration, but the convulsions became more frequent and severe, and the patient died in twenty-five minutes without becoming conscious. No necropsy was performed.

CASE 15.—A woman aged 35 had been receiving artificial pneumothorax therapy for three weeks. On the fifth treatment the manometer showed a negative pressure of —8 cm. of water. After 125 cc. of air had been induced, the patient became pale and unconscious. The needle was withdrawn and the end was blood tinged. Generalized clonic convulsions followed; the patient became cyanotic and swallowed her tongue. The pulse was imperceptible, the respiration gasping and convulsive. Epinephrine, ammonia and artificial respiration were given. The patient became conscious in fifteen minutes but complained of blurred vision and severe headaches for three days.

CASE 16.—A woman aged 28 was receiving artificial pneumothorax on admission to the sanatorium in May 1926. On the seventh pneumothorax treatment in the sanatorium she suddenly became unconscious after 100 cc. of air had been given. The needle was withdrawn and the tip was blood tinged. The patient was limp and pale, and no pulse was palpable. The respiration was irregular. Epinephrine and artificial respiration were given. The patient became conscious in ten minutes but complained of exhaustion and nausea for the next twelve hours.

CASE 17.—A white woman aged 27 had a tuberculous empyema. During aspiration and irrigation of the empyema cavity she suddenly complained of weakness, vertigo and severe pain over the precordium and down the left arm. She was semiconscious for about five minutes and uttered loud groaning noises. For twenty-four hours she complained of precordial pains, pain and numbness in the left arm and blurred vision.

TREATMENT

Treatment of air embolism consists in prophylactic and therapeutic measures. Prophylaxis deals mainly with the technic employed in giving artificial pneumothorax treatments. A thorough study by roentgen ray and fluoroscopic examinations will show areas of consolidation, adhesions and cavitation near the periphery of the lung and will guide the operator away from these dangerous areas. The pneumothorax needle should be introduced attached to a syringe of procaine hydrochloride. In this way, once the needle has penetrated the parietal pleura, air can be drawn back freely into the syringe whereas, if the needle has penetrated the lung or an adhesion or is in a blood vessel, on retraction of the syringe plunger frothy blood tingled fluid or blood will be withdrawn. If air bubbles obtained are from the lung they are usually blood tinged and difficult to withdraw. Unless the needle aspirates air freely it should be withdrawn immediately and another puncture site selected. The introduction of the needle should be direct and with as little manipulation as possible.

During refills, free manometer oscillations should be obtained before air is introduced.

If the patient complains of severe pain, nausea or vertigo or other symptoms common to air embolism, the needle should be withdrawn and the patient's head lowered for at least ten minutes.

Once air embolism has occurred, the quicker the air bubbles can be removed from the coronary and cerebral circulation the better the prognosis. Small air bubbles are absorbed spontaneously in a period of ten to twenty minutes, and, if the patient can be kept alive by artificial respiration and stimulants until absorption of the air occurs, recovery will follow. The head should be lowered to an angle of 30 degrees to prevent further cerebral embolism. Epinephrine hydrochloride and caffeine with sodium benzoate are circulatory and respiratory stimulants and aid the circulation in dispersing the air bubbles.

In the event of impending respiratory failure, artificial respiration is necessary.

The head of the patient's bed should be kept lowered for at least half an hour after cessation of symptoms.

Carbon dioxide [17] has been advocated as a substitute for air in the initial and first few pneumothorax treatments. It is absorbed much more rapidly than air and consequently is of less danger if embolism occurs.

SUMMARY

1. Of the 17 cases reported, 10 cases—2, 4, 5, 6, 7, 8, 10, 11, 13 and 14—were apparently due to cerebral air embolism. These were characterized by pallor, partial or complete loss of consciousness, and residual headaches and blurred vision. Among these cases there were 4 fatalities.

2. Five cases—1, 3, 9, 12 and 16—were apparently due to coronary air embolism and presented the symptoms of unconsciousness, cyanosis, irregular heart action, absence of convulsive seizures and presence of convulsive gasping respiratory movements. Three of the 5 patients died.

3. Two cases—15 and 17—presented symptoms of both coronary and cerebral embolism. Neither reaction was fatal.

4. In 9 cases the reaction occurred during the process of inducing air, in 4 cases after the needle was removed, in 3 cases before air was given and in 1 case during the aspiration of pleural fluid.

5. The amount of air induced before the reaction varied from none to 300 cc.

6. The number of air emboli on the various pneumothorax treatments was as follows: one on the first, four on the third, two on the fifth, one on the seventh, two on the tenth, one on the eleventh, one on the fifteenth, one on the eighteenth, three after many treatments, and one during aspiration of an empyema cavity. Half of the air emboli occurred on or before the tenth pneumothorax treatment.

7. The symptoms and signs of air embolism are pallor, dizziness, blurred vision, papillary dilatation and failure to react, ocular deviation, nausea, unconsciousness, tonic and clonic convulsions, mottling of the skin, air bubbles in retinal vessels, paresthesia, precordial pain, irregular respiration, abnormal heart action and residual headache.

8. Clinically, cerebral air embolism seems to occur more often than coronary embolism, but in 35 cats air induced in the pulmonary vein caused death by coronary occlusion without exception.

9. Among the 17 patients who had air embolisms, 11 had adhesions and poor compressions, 5 had good compressions, and 1 reaction occurred on the initial puncture.

17. Moore, R. M., and Braselton, C. W.: An Experimental Study of the Embolic Effects of Air and of Carbon Dioxide, South. Surgeon 9: 733 (Oct.) 1940.

CONCLUSIONS

1. Air embolism occurs more frequently in patients with adhesions and poor compressions. Half occur during the first ten pneumothorax treatments.

2. An air embolus occurs once on an average among every 79 pneumothorax patients, an incidence of 1.3 per cent, and once among every 2,104 pneumothorax treatments, an incidence of 0.047 per cent.

3. The mortality from air embolism is about 29 per cent.

4. The treatment consists in lowering the patient's head and giving artificial respiration and stimulants, as epinephrine hydrochloride and caffeine with sodium benzoate.

Clinical Notes, Suggestions and New Instruments

A DEFINITE AND UNIQUE OCCURRENCE OF RAPIDLY FATAL INFECTION CAUSED BY BACILLUS VIOLACEUS MANILAE

HERBERT J. SCHATTENBERG, M.D., AND WILLIAM H. HARRIS, M.D., NEW ORLEANS

Considerable skepticism is extant regarding the pathogenicity of Bacillus violaceus. It has been generally considered as a nonpathogenic saprophyte having a rather ubiquitous distribution. More careful perusal and consideration of the sparse literature pertinent to this subject reveals, however, that much of the doubt is due to failure of recognition of the fact that many distinctive species exist in the group of which Chromobacterium violaceum is the type species. For a comprehensive classification of this group as regards both the cultural and the pathogenic aspect, the reader is referred to the presentation of Ford.[1]

Woolley [2] identified by pathogenicity and by other specific biologic reactions a species of this group producing fulminating fatal infections in the caribao, or water buffalo, which he termed B. violaceus manilae.

In the 2 human cases recorded, one by Black and Shahan [3] and the other by Soule,[4] the durations of illness were of nine and fifteen months respectively. In the report of Black and Shahan the patient had seemingly recovered after approximately two months but in the later report of Soule it is stated that the patient of Black and Shahan died after about nine months. The patient of Soule was sick for approximately fifteen months and, although the micro-organism had been obtained in culture from the cervical adenitis present, it was not considered as the cause of the illness until necropsy. B. violaceus was then recovered from the heart's blood and the lesions present in the various organs, and its etiologic role appeared established.

The causative micro-organism concerned in the present report conformed in all respects to B. violaceus manilae. Certain experimental aspects of the results obtained have already been reported [5] and the subject matter in extenso is also to be published.

In the present case there occurred a definite invasion of the micro-organism with resultant septicemia and ensuing death within a week. No other factors were present whereby confusion or doubt could arise relative to the causation of illness and death.

Aided by a grant from the David Trautman Schwartz Research Fund.
From the Department of Pathology of Tulane University School of Medicine and the Tulane Service of the Charity Hospital of Louisiana.

1. Ford, William W.: Textbook of Bacteriology, Philadelphia, W. B. Saunders Company, 1927, p. 468.
2. Woolley, P. G.: Bull. Johns Hopkins Hosp. **16:** 89, 1905.
3. Black, M. E., and Shahan, John: Bacillus Violaceus Infection in a Human Being, J. A. M. A. **110:** 1270 (April 16) 1938.
4. Soule, M. H.: Am. J. Path. **15:** 592 (Sept.) 1939.
5. Schattenberg, H. J.: Proc. Soc. Exper. Biol. & Med. **45:** 829 (Dec.) 1940. Schattenberg, H. J., and Brown, D. V., ibid. **46:** 478 (March) 1941.

REPORT OF CASE

C. M., a Negro youth aged 21 years, admitted to Charity Hospital on Aug. 31, 1940, was lethargic and incoherent, so that no satisfactory history was obtainable from him. His sister stated that on Aug. 29, 1940 it was noticed that he was limping on his right foot and it was thought that he had a thorn in it. She said that he had attempted to remove the thorn with his knife. When he arose the next morning he complained of headache and of pain in the right leg. He appeared "feverish" and after a hard chill he was brought to the hospital.

The patient was well developed and nourished. He was delirious and in moderate pain. The temperature was 104.2 F., the pulse rate 112, the respiratory rate 36 and the blood pressure 86 systolic and 72 diastolic. Examination of the head, neck, chest, abdomen and external genitalia showed nothing abnormal. A small abrasion was noted on the sole of the right foot with accompanying tenderness of the entire right leg. There was diffuse tenderness in the right inguinal region, wherein enlarged, firm, nodular inguinal lymph nodes were palpable. Blood counts showed 3.92 million red blood cells and 26,900 white blood cells. Urinalysis and the blood Wassermann reaction were negative. Blood urea, uric acid and dextrose were within normal limits. Blood cultures were taken which after four days yielded heavy growths of B. violaceus. Blood agglutinations for tularemia, typhoid, paratyphoid and bacillary dysentery were negative. During the five days in the hospital the patient's temperature ran between 104 and 105 F. excepting the day before death, when the temperature dropped to 102.5 F. The pulse rate varied from 112 to 140 and the respiratory rate from 22 to 60.

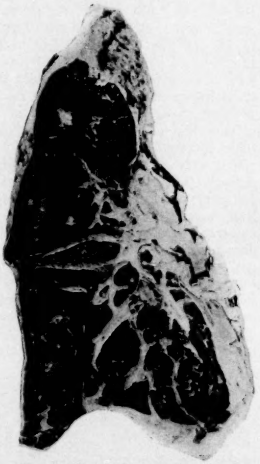


Fig. 1.—Longitudinal section of lung showing scattered foci of necrosis of irregular shape and size, more numerous in the upper lobe.

On the fourth day after admission to the hospital the patient had signs of diffusely consolidated areas in the lungs and expectorated blood tinged sputum. His scleras became icteric. The terminal picture was one of pyrexia and circulatory collapse ending in death seven days after onset of symptoms and five days after admission to the hospital.

Treatment consisted of the administration of tetanus antitoxin, sulfanilamide, infusions of 10 per cent dextrose solution, transfusions of citrated whole blood, sedation, fluids and oxygen.

At necropsy the body measured 175 cm. in length and weighed approximately 180 pounds (81 Kg.). The pupils were round and equal and measured 6 mm. in diameter. The scleras were deeply icteric. Extensive dental caries was seen, with evidence of poor oral hygiene. No unusual cervical or axillary adenopathy was noted. The thorax and abdomen were normal. No scars or lesions of any sort were seen about the external genitalia or perineum. The inguinal lymph nodes on the right side were enlarged, firm and discrete; the nodes on the left showed no gross abnormalities. Both lower extremities were symmetrical, and no edema or swelling could be detected. On the ball of the right foot, just posterior to the fifth metatarsophalangeal junction, there was a superficial area of denudation

of the cornified epithelium measuring 2 by 2 cm. No especial inflammatory reaction was present surrounding this area. The abdominal cavity presented no gross abnormalities. The pleural cavities contained 20 to 25 cc. of serosanguineous fluid. There were numerous firm fibrous adhesions on the right side between the parietal and visceral pleurae. The heart weighed 340 Gm.

Fig. 2.—Sectioned surface of liver in which are seen four white foci of necrosis.

The myocardium was of good tone throughout. The heart valves were normal. The right lung weighed 840 Gm., the left 640 Gm. Just beneath the pleural surfaces of both lungs were numerous elevated yellowish areas measuring from 2 mm. to 1.5 cm. (fig. 1). On palpation these areas were found to consist of irregularly rounded firm nodules. Such nodules were likewise felt deeply throughout all lobes. On cut section these lesions stood out from the normal lung tissue and on pressure exuded a small amount of milky fluid. No areas of cavitation or liquefaction were noted. The bases of both lungs appeared to be the most heavily involved portions. The spleen weighed 500 Gm. and was of a uniformly firm consistency. On cut section the pulp was found to be dark red and very friable; it scraped away easily on the knife. The liver weighed 1,920 Gm. Scattered over its surface were numerous oval yellowish areas varying from 1 to 5 mm. in diameter. On sectioning through the liver substance numerous similar lesions were found deeper in the organ (fig. 2), some of which appeared to have undergone softening with formation of a thin creamy brown fluid. A few areas as large as 1 cm. in diameter were seen which were yellowish and of increased firmness but somewhat poorly demarcated. The pancreas weighed 160 Gm. and appeared normal. The gastrointestinal tract presented no gross abnormalities. The right kidney weighed 140 Gm., the left kidney 170 Gm. Both showed only slight swelling and congestion grossly. The brain presented nothing noteworthy on gross inspection. On dissection and inspection of the inguinal lymph nodes on the right it was observed that they measured from 0.5 to 4 cm. in diameter. They were fairly discrete and were but slightly adherent to the surrounding structures. They were dark gray, and on palpation evidence of fluctuation could be detected. On section a small amount of dark gray purulent material was found in the necrotic centers of the lymph nodes. The surrounding tissues, including the femoral vein, showed no extension of the inflammatory process.

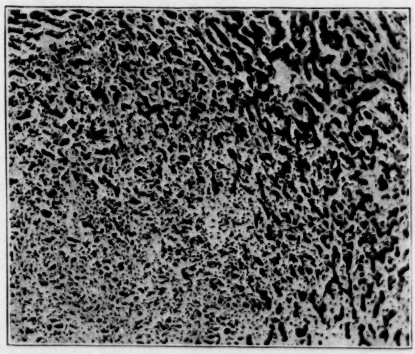


Fig. 3.—Section of peripheral portion of necrotic focus in liver shown in lower left portion. Extensive necrosis of liver cells are shown with but little cellular reaction.

Cultures were procured from the lesions of the lung, liver, inguinal lymph nodes and the spleen.

The anatomic diagnosis was Chromobacterium violaceum bacteremia with abscess formation in liver, lungs and spleen; bronchopneumonia; acute hepatitis; acute splenitis; acute lymphadenitis.

Bacteriologic examination revealed growths of B. violaceus in all cultures. These were eventually identified as B. violaceus manilae (Woolley).

In general, the microscopic study of sections of the lesions revealed analogous pathologic changes. In many of these, necrosis with little or no cellular response was present (fig. 3). Other foci consisted in greater part of necrosis with varying degrees of cellular and fluid exudate. Serum, red blood cells, neutrophils and lymphoid cells of different amounts were present. In the lungs extensive edema and some hemorrhagic extravasation surrounded the necrotic foci (fig. 4).

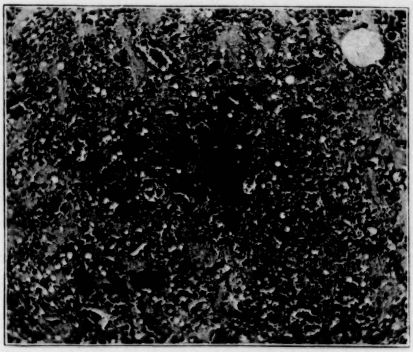


Fig. 4.—Section of lung revealing a central focus of necrosis with surrounding edema of the alveoli.

COMMENT

While much doubt has heretofore existed as to the pathogenicity of B. violaceus for the human subject, a review of the present case reveals a definite fulminant, fatal infection caused by this micro-organism. The facts presented are an illness of brief duration with a history of likely exposure to infection, a septicemia with positive blood culture, resultant death and recovery of the micro-organism from the visceral lesions. It is also to be noted that no associated source of confusion by other infection or disease existed.

The proved pathogenicity for lower animals both by injection and by experimental splinter routes of infection have previously been reported.[5]

The continuity of clinical and experimental evidence supplied through the present case forms an incontrovertible sequence of fatal human infection with B. violaceus manilae.

We wish to stress the fact that there exists a rather universal acceptance of the predominantly saprophytic varieties of this group with little or no recognition that at least one species is of proved pathogenic nature, namely B. violaceus manilae (Woolley). Because of this fact it appears probable that when this micro-organism is occasionally recovered in certain infections, although truly the causative factor, it is unjustifiably disregarded.

TULAREMIA IN DOGS

L. F. EY AND R. E. DANIELS, COLUMBUS, OHIO

From the literature on the subject of tularemia we realize that some questions remain undetermined in regard to the susceptibility of certain animals and birds to this disease. For example, it is evident that sufficient evidence has not been produced to place the common dog in the group of susceptible or naturally immune animals. Obviously it can be understood that the material required to study the susceptibility of dogs under natural conditions is not always readily accessible. Consequently investigators interested in this phase of the problem have limited their observations to experimental studies. From the few experimental studies we are given the impression that the dog is only mildly susceptible to tularemia, and other opinions indicate that this animal possesses a natural immunity.

In this communication we are prepared to show that dogs can contract tularemia under favorable natural conditions and that a resultant high agglutination titer for Bacterium tularense can be observed in the blood serum following a comparatively short duration of the disease.

Dr. Edward Francis[1] in all probability carried out the original experimental studies on dogs. In 1927 he fed infected guinea pig tissues to two dogs, one for twenty-one days and the other for twenty-five days. On the forty-second day he performed agglutination tests on the blood serum of the dog fed for twenty-one days and the serum showed a response in a dilution of 1:320. The blood serum of the dog fed for twenty-five days was subjected to agglutination tests after the fifty-third day, and the reaction was positive to a titer of 1:640. Further similar experimental studies were made by Francis in 1934 on 4 dogs that were injected with virulent cultures, 1 of which died of tularemia after fourteen days but the other 3 remained well. The 3 developed antitularense agglutinins of 1:160, 1:640 and 1:1,280 respectively. It is his belief that a dog could not be killed by being fed infected tularemic material but that by enormous dosages of the organism injected subcutaneously an occasional dog will die. He concluded that dogs are only slightly susceptible to tularemia.

The cases in dogs which prompted our investigations were brought to our attention in connection with a human case of tularemia. During a conversation with the patient's brother-in-law on Nov. 15, 1940, one of us (L. F. E.) learned that 3 dogs went hunting with Mr. G. D. and that there was a possibility that the dogs might also be afflicted with tularemia. Our informant minimized the significance of their condition, but the lead gave us the opportunity of investigating the cause of sickness. We wish, therefore, to touch on several salient points of the human case.

REPORT OF CASE

Mr. G. D., a patient of Dr. G. O. Thompson of Alliance, Ohio, went hunting on Nov. 6, 1940, with his 3 dogs. The dogs ran down a rabbit, which they killed. Mr. D. picked up the dead rabbit, tore it in three pieces and gave each dog a portion. The following day, November 7, one of the dogs, the youngest, became ill and on November 8 Mr. D took this dog to a local veterinarian Dr. George F. Nixon.[2]

While talking with Dr. Nixon concerning the symptoms of the dogs, Mr. D. expressed the thought that perhaps they had contracted "rabbit fever." Apparently at that time Mr. D. felt normal, or at least he was not aware of having contracted the same infection. According to Dr. Nixon, the 2 other dogs became ill a day or two later and, while none were hospitalized, on examination he found no elevation of temperature but described them as being more or less listless, with little or no appetite.

On November 9, three days after handling the rabbit, Mr. D. became quite ill with chills and fever and on November 11 he was hospitalized. In the hospital when informed that the 2 other dogs were also showing signs of illness, he remarked "Well, that is strange. That rabbit didn't run like a healthy one; maybe it was sick."

From consultant Dr. Perry F. King's record in the hospital, it is easily recognized that Mr. D. was a victim of tularemia, and, as one might expect, the site of his initial infection occurred on the hands. The right hand in this instance showed two lesions of the skin, of a bluish red discoloration. One lesion on the index finger appeared as a large papule.

Blood collected from Mr. D. on November 12 was submitted to the Laboratory Division, Ohio Department of Health, with a request that it be examined for typhoid and undulant fever. Routine agglutination tests for these infections gave negative results. A second specimen taken on November 15, nine days from the date of exposure or six days following the onset of the initial symptoms, was forwarded to us for Pasteurella tularensis agglutination test. The blood serum reaction was negative, as were repeated tests for typhoid and Brucella abortus.

Although the patient received antitularense serum treatment, he died on the night of November 16, seven days from the date of onset or ten days after exposure to the infection. This, of course, is regarded as a short duration of illness and unquestionably accounts for the failure to obtain a positive Past. tularensis agglutination reaction. In other words, while the degree of infection was overwhelming, the time was too short for the development of serum agglutinins.

This case, when considered with respect to the short duration of illness and death, is not without a parallel experience, for it is a matter of record that in 1928 Simpson[3] reported a case of tularemia which terminated fatally four days and seven hours after the onset of the illness.

On the information supplied by the patient's brother-in-law we laid plans to obtain blood specimens from the 3 suspected dogs. Dr. Nixon collected the blood and we received the specimens on November 25, nineteen days after the date on which the dogs ate the infected rabbit. It was interesting to note that 4 plus agglutination with Past. tularensis antigen was observed in each serum beginning with dilutions of 1:20 to 1:320 inclusive, and 3 plus readings in 1:640. Agglutination was absent in a titer of 1:1,240.

In order to rule out the possibility of nonspecific agglutinations in these dogs' serums, plans were carried out to obtain serum from a sufficient number of presumably normal dogs for control purposes. Dr. Nixon submitted the first control series of 7 specimens between December 2 and 9, all of which were taken from dogs in his hospital. These gave no indication of agglutination response. About the same time we consulted Dr. W. H. Hobbs, of the College of Veterinary Medicine, Ohio State University, for additional control material, and he consented to furnish us with three important series of blood specimens. We emphasized the importance of procuring specimens from dogs owned by hunters or farmers. Our object in making this request was to examine blood from dogs which had the closest contact with game animals, including ticks and fleas. In December 1940, Dr. Hobbs supplied 14 blood specimens and in February 1941 we received 10, a total of 24 specimens. The ages of the dogs ranged from 1½ to 10 years, and approximately twelve different breeds were represented in the group. Six were definitely listed as hunting dogs, 6 were owned by persons living in large cities and 12 belonged to farmers or residents in small towns.

All the control serums were subjected to the same procedure as was the serum in the 3 positive cases, but no evidence of agglutination for Past. tularensis was observed in any one of the 24 control specimens furnished by Dr. Hobbs.

At the time the results of the tests on the original 3 dogs were reported to Dr. Nixon it was pointed out that subsequent blood examinations would be essential in order further to substantiate our observations and to learn particularly whether the titer of the serums would show a reduction similar to those found in human cases after a lapse of several months.

From the Laboratory Division, Ohio Department of Health.

1. Francis, Edward: Personal communication to L. F. E.

2. It was largely through the cooperation received from Dr. Nixon that we were able to carry on the studies presented in this paper, since Dr. Nixon furnished us with the records of the dogs and several series of blood specimens including blood specimens from normal dogs for control purposes.

3. Simpson, W. M.: Tularemia: Study of Rapidly Fatal Case (Four Days, Seven Hours), Arch. Path. 6: 553 (Oct.) 1928.

As one of the concluding steps in this study, Dr. Nixon was asked to collect a second set of specimens from these 3 dogs. On March 28, 1941 we had the opportunity of making the confirmatory observations on these specimens, and, as was to be expected, there was a diminished titer noted in each serum. While the original tests were positive in dilution of 1:640, the highest dilution in which agglutination occurred was 1:80, 2 showing a 1 plus reaction and the third gave no reaction. However, in dilutions of 1:20 and 1:40, 3 plus readings were recorded in all.

The dogs recovered and are alive today. Periodic rechecking of these animals is, of course, highly desirable, and provided the dogs are available we intend to make additional tests of blood specimens.

SUMMARY AND CONCLUSION

1. Evidence is presented to show that dogs may naturally acquire mild tularemia by the ingestion of infected rabbit material.

2. Dogs so infected will develop Pasteurella tularensis agglutinins in high titer in less than twenty days.

3. It is suggested that blood specimens from dogs manifesting potential Past. tularensis infection be subjected to routine agglutination tests.

4. A man died of tularemia seven days after the onset of illness. The man and his 3 dogs acquired the infection from the same rabbit. The dogs survived.

Council on Physical Therapy

THE COUNCIL ON PHYSICAL THERAPY HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORTS. HOWARD A. CARTER, Secretary.

TELEX HEARING AID, MODEL 1020, ACCEPTABLE

Manufacturer: Telex Products Company, 1645 Hennepin Avenue, Minneapolis.

The Telex Hearing Aid, Model 1020, is said by the manufacturer to be essentially the same as the Council accepted Model 612 except for a change in the case.

Telex Hearing Aid, Model 1020.

In the Council examination the device was found to consist of one microphone and amplifier unit, one crystal receiver, one 30 volt B battery W. 20, one 1.5 volt A battery 2 E. S. and one leather battery case. The weight and dimensions of the various parts are given in table 1.

BATTERY DRAIN

The A battery current shown was 90 milliamperes, independent of the setting of the volume control. The B battery current was independent of the volume control setting but showed a variation between 0.5 and 1.0 milliampere, depending on the sound intensity being picked up by the microphone.

TABLE 1.—*Weight and Dimensions*

	Dimensions, Inches	Weight, Ounces
Microphone and amplifier unit	4¼ by 2¼ by ¾	3.86
Receiver	Diameter 1, thickness ⅜ inch	0.2 (without earpiece)
Battery case with batteries	4½ by 2½ by 1¼	11.8

INTERNAL NOISE

The internal noise is not excessively high. The instrument is provided with an auxiliary gain control, with three settings for controlling feed-back. Using a well fitted earpiece, the volume could be set as high as three fourths of full volume without feed-back when the auxiliary control was set on contacts 1 and 2. Set on contact 3, the volume could be turned full on without feed-back. Acoustical gains were measured with these last named settings of the controls.

ACOUSTICAL GAIN

The figures given in table 2 are typical of the increase in intensity levels at the ear of the subject over that at the microphone as shown by audiometric measurements.

TABLE 2.—*Increase in Intensity Levels*

	Normal Ear Threshold at Microphone	Gain 256-2,048		4,096
		Min.	Max.	
Volume setting	128			
Full on	None	13	43	14 decibels
	40 Decibels Above Normal Ear Threshold			
Full on	?	15	44	25 decibels

ARTICULATION

Syllable and sentence tests with hard of hearing subjects gave satisfactory results.

The Council voted to accept the Telex Hearing Aid, Model 1020, for inclusion on the list of accepted devices.

O. E. M. METER MASK ACCEPTABLE

Manufacturer: Oxygen Equipment Manufacturing Company, Inc., 405 East Sixty-Second Street, New York.

The O. E. M. Meter Mask consists of a gum rubber face piece, nasal or oronasal, adjustable head bands, an expiratory flutter valve, a latex collecting bag, an inspiratory check valve, an oxygen percentage meter, a single stage regulator and 4½ feet of rubber tubing. Inhaled atmospheres enter from the latex collecting bag and directly from the supply tubing, past a one way mica flutter valve, thence via the two lead-in tubes of the nasal masks, or through the one lead-in in the case of the oronasal mask. Exhaled atmosphere cannot reenter the collecting bag but leaves through the flutter valve in front of the nose.

O. E. M. Meter Mask.

Oxygen from cylinders or other sources passes first through a small metal chamber, which may be kept entirely closed or partly opened by turning a disk in which are holes of graded size labeled 40, 45, 50, 60, 70, 80, 90 and one position with no hole, marked 95+. These are designed to draw varying amounts of air along with the oxygen as it flows through a jet into the tube supplying the face piece, or no air when the 95+ position is used, closing the metal chamber entirely.

In the Council's examination the apparatus was subjected to the following tests:

A. The nasal mask was worn by a normal human subject for thirty minutes with oxygen flowing at a rate sufficient to keep the collecting bag from collapsing completely on inspiration (7.5 and 8.5 liters per minute) and with the meter disk set at 95+. At the end of this time, through a tube piercing the mask just below the nostrils, a large sample was drawn by several small aspirations at midinspiration. This sample was then analyzed for carbon dioxide and oxygen concentrations by the manometric method of Van Slyke and Peters.

This procedure was repeated, using an oxygen flow of 7 liters per minute with the meter disk set at 90, with 6 liters per minute and disk setting of 80, with 4.3-4.5 liters per minute and disk setting of 60, with 3-3.4 liters per minute and disk setting of 50, and with 2.4 liters per minute and disk setting

of 40. (In each of these tests a large flow of oxygen with the meter disk set at 95 + was first employed for five to ten minutes to wash out the free nitrogen in the lungs before sampling under the conditions just stated.)

B. Since the oronasal mask could differ from the nasal only in the amount of dead space and rebreathing, it was tested similarly for carbon dioxide concentrations only.

C. The fit of the mask about the nose and mouth was investigated by trial and by holding an open bottle of strong ammonia solution closely below the face piece and having the subject inhale deeply.

D. The face piece with exhalation flutter valve and the supply tubing were boiled for three minutes to test the durability of the seal of the valve parts. These and other parts were subjected to prolonged immersion in red mercuric iodide solution, commonly used for short periods for sterilization of nonboilable catheters.

Following are the results of the foregoing tests:

A. NASAL MASK

Meter Disk Setting	Oxygen in Liters per Minute	Oxygen, per Cent	Carbon Dioxide, per Cent
95+	7.5	87.3	0.076
	8.5	96.8	0.000
90	7.0	67.8	0.000
	7.1	63.5	0.400
	7.0	60.8	0.420
80	6.0	69.4	.....
	6.0	74.9	0.260
70 (not tested)			
60	4.5	44.6	0.470
	4.3	39.0	.....
50	3	42.3	0.073
	3.4	38.3	0.240
40	2.4	42.8	0.000

B. ORONASAL MASK

Meter Disk Setting	Oxygen in Liters per Minute	Carbon Dioxide, per Cent
95+	8.5	0.06
60	4.5	0.26

C. The masks were easy to fit comfortably for prolonged periods when the head straps passed below the ears. No leaks were detected with the strong ammonia vapor.

B. Boiling the face piece for three minutes did not damage it or the exhalation valve. Prolonged immersion in red mercuric iodide did not harm the flutter valve or any other parts.

The Council voted to accept the O. E. M. Meter Mask for inclusion on its list of accepted devices.

Council on Pharmacy and Chemistry

REPORT OF THE COUNCIL

THE COUNCIL HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORT.

THEODORE G. KLUMPP, M.D., Secretary.

ANTIPNEUMOCOCCIC SERUM (RABBIT) TYPE XIV

The Council has accepted types I, II, IV, V, VII, VIII of antipneumococcic horse serum and types I, II, III, IV, V, VII, VIII of antipneumococcic rabbit serum. Recently one manufacturer (E. R. Squibb & Sons) presented Antipneumococcic Rabbit Serum, Type XIV, and, after reviewing the situation, the Council decided that sufficient evidence now exists to warrant the acceptance of this type. The status of the other types of antipneumococcic rabbit serums, on the clinical use of which experience is accumulating, is at present under review and will be made the subject of a Council report.

NEW AND NONOFFICIAL REMEDIES

THE FOLLOWING ADDITIONAL ARTICLES HAVE BEEN ACCEPTED AS CONFORMING TO THE RULES OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR ADMISSION TO NEW AND NONOFFICIAL REMEDIES. A COPY OF THE RULES ON WHICH THE COUNCIL BASES ITS ACTION WILL BE SENT ON APPLICATION.

THEODORE G. KLUMPP, M.D., Secretary.

QUININE DIHYDROCHLORIDE.—For standards see the U. S. Pharmacopeia under Quininae Dihydrochloridum.

Actions and Uses.—Quinine Dihydrochloride has actions similar to those of quinine, over which it has the advantage of being more soluble in water. It is used where aqueous solutions of quinine are desired, as for parenteral injections.

Intramuscular and especially intravenous administration of solutions of quinine dihydrochloride should be reserved for cases of severe malarial infection in which oral medication is not feasible. In those cases in which intravenous administration is deemed necessary this solution must be diluted to a concentration not greater than 0.5 per cent.

Dosage.—From 0.24 to 1 Gm. (3¾ to 15½ grains) intramuscularly or intravenously as indicated by the severity of the symptoms, age and the like.

The Lakeside Laboratories, Inc., Milwaukee.

Ampuls Quinine Dihydrochloride 0.24 Gm. (3¾ grains) in 1 cc.; 0.49 Gm. (7½ grains) in 1 cc.; 1.0 Gm. (15½ grains) in 2 cc. (For Intramuscular Use): Each ampul contains the stated amount of quinine dihydrochloride dissolved in distilled water.

Ampuls Quinine Dihydrochloride 0.32 Gm. (5 grains) in 5 cc.; 0.49 Gm. (7½ grains) in 5 cc.; 0.49 Gm. (7½ grains) in 10 cc.; 0.65 Gm. (10 grains) in 20 cc. (For Intravenous Use): Each ampul contains the stated amount of quinine dihydrochloride dissolved in distilled water.

ANTIPNEUMOCOCCIC SERUM, TYPE XIV (FROM RABBITS).—An antiserum obtained from the blood of an animal of the genus Lepus, containing predominantly antibodies for type XIV pneumococcus (Diplococcus pneumoniae).

Dosage.—Average dose, 20,000 to 100,000 units intravenously. The initial and subsequent doses should be administered in such amount and at such intervals as indicated by the condition of the patient, according to the judgment of the physician. With a large initial dose, fewer units may be curative. The Francis cutaneous test (injection of specific pneumococcic capsular polysaccharide) may be used in conjunction with clinical observations to determine adequacy of dosage.

E. R. Squibb & Sons, New York.

Antipneumococcic Rabbit Serum, Type XIV.—It is refined and concentrated by salting out with ammonium sulfate, the globulin fraction being retained. The final product contains merthiolate 1 in 10,000 and 0.2 per cent of phenol. Marketed in vials containing 20,000 units and 50,000 units each. Included in the trade package is a 5 cc. vial containing 0.05 cc. of the same concentrated rabbit serum diluted in 5 cc. of physiological solution of sodium chloride for the sensitivity test.

THIAMINE HYDROCHLORIDE-SQUIBB (See New and Nonofficial Remedies, 1941, p. 551, under Thiamine Hydrochloride).

The following additional dosage forms have been accepted:

Crystals Thiamine Hydrochloride-Squibb: Marketed in bottles containing 1 Gm. of crystalline thiamine hydrochloride, equivalent to 333,333 U. S. P. units.

Solution Thiamine Hydrochloride-Squibb, 10 mg. per cc., and 50 mg. per 25 cc. vials: Each cc. contains in addition 5 mg. of chlorobutanol in sterile distilled water.

NICOTINIC ACID AMIDE (See New and Nonofficial Remedies, 1941, p. 556).

The following dosage form has been accepted:

Ampuls Nicotinic Acid Amide-Breon, 50 mg. in 2 cc.: Each 2 cc. contains 50 mg. of nicotinic acid amide in physiological solution of sodium chloride.

Prepared by George A. Breon & Co., Inc., Kansas City, Mo. No U. S. patent or trademark.

NICOTINIC ACID-U. S. P. (See New and Nonofficial Remedies, 1941, p. 555).

The following dosage forms have been accepted:

Ampoules Solution Nicotinic Acid, 10 mg. in Physiological Sodium Chloride Solution, 10 cc.

Prepared by Endo Products, Inc., Richmond Hill, N. Y.

Ampoules Solution Nicotinic Acid, 20 mg. in Physiological Sodium Chloride Solution, 10 cc.

Prepared by Endo Products, Inc., Richmond Hill, N. Y.

Ampoules Solution Nicotinic Acid, 100 mg. in Physiological Sodium Chloride Solution, 10 cc.

Prepared by Endo Products, Inc., Richmond Hill, N. Y.

DEXTROSE (See New and Nonofficial Remedies, 1941, p. 179).

George A. Breon & Co., Inc., Kansas City, Mo.

Ampules Solution Dextrose 50% (W/V), 50 cc.: Each ampule contains 50 cc. of a solution containing 25 Gm. of dextrose, U. S. P.

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

535 NORTH DEARBORN STREET - - - CHICAGO, ILL.

Cable Address - - - - "Medic, Chicago"

Subscription price - - - - - - Eight dollars per annum in advance

Please send us promptly notice of change of address, giving both old and new; always state whether the change is temporary or permanent. Such notice should mention all journals received from this office. Important information regarding contributions will be found on second advertising page following reading matter.

SATURDAY, DECEMBER 13, 1941

THE NATION AT WAR

On December 7 the Japanese attacked the United States. On December 8 the President declared a state of war between Japan and the United States; both the Congress and the Senate of the United States adopted resolutions supporting the action of the President. The mustering of the forces of the United States to meet this attack and to carry on warfare to absolute victory places an immediate and tremendous responsibility on the medical profession. By the time this editorial appears, the Selective Service will, no doubt, have intensified its activities to call an additional half million, if not a million, men. For every thousand men called into service, at least six physicians are required.

Fortunately the medical profession has been alert. Since June 1940 the medical profession has been intensively engaged in standardization of military medical procedures, encouragement and promotion of scientific military medical research and enrolment of medical personnel. Already more than twenty-five thousand physicians have given of their services to the Selective Service. Already more than ten thousand physicians are engaged in military medical service. Additional thousands are associated with the United States Army and Navy Medical Corps, the United States Public Health Service, speeded industry and many other military and quasimilitary activities. Moreover, the Office of Civilian Defense has made the medical profession aware of requirements for its services to the civilian population in times of emergency.

Whenever the medical profession has been called on by our government it has responded whole-heartedly and enthusiastically to the Nation's call. Never has there been raised the slightest doubt of the patriotism of the medical profession. Now comes a new opportunity to respond.

The development of the Procurement and Assignment Service for Physicians, Dentists and Veterinarians places on the medical profession the responsibility for supplying the medical personnel necessary to all the services that have been mentioned. The utilization of the roster in the headquarters of the American Medical Association gives opportunity to supply all the needed officers and, at the same time, to protect the requirements of medical education and the care of the civilian population.

On December 18 the Committee on Medical Preparedness of the American Medical Association will meet in Chicago together with similar committees representing the dental and veterinarian professions to outline the details of the method by which physicians will be called. The state and county committees on medical preparedness will be utilized in developing complete information regarding physicians available for various types of service. Shortly also THE JOURNAL will publish a blank on which every physician willing to serve may indicate the special service for which he is immediately available.

The organization has been made ready; the lines of communication are drawn; confusion need not prevail. When the call comes, physicians who are to serve with the military branches will enroll directly through the corps area commander in each corps area and all of the necessary steps will have been taken to facilitate such enrolment. The President of the United States will find the physicians of this nation ready and willing to meet any demands which the nation may place on them.

THE POTENCY OF DIGITALIS

Elsewhere in this issue (Correspondence, p. 2093) appears a communication from the chairman of the Subcommittee on Biological Assays of the United States Pharmacopeia calling attention to the changes in strength and in method of assay of the digitalis preparations of the next revision of the United States Pharmacopeia. Important to the physician is recognition of these changes and understanding of their significance.

Justification for standardization of medicines lies in the facilitation of the interchange of experience. In successive revisions of the United States Pharmacopeia in past decades certain potent drug preparations have been made to conform to international practice. In the revision now current, the eleventh, effective since June 1, 1936, the strengths of powdered digitalis and tincture of digitalis have been adjusted to conform to the standards of the League of Nations. Those responsible for this decision knew that an increase in the strength of digitalis in use in the United States would result. However, the information apparently did not reach the medical profession generally. Only when experience began to reveal the change did it get the proper publicity. Unfortunately by this time there had developed a controversy over what is to some extent a secondary matter. How much had the strength been increased? The chairman of the Subcommittee on Biological Assays of the Eleventh Revision firmly believed that the increase was about 30 per cent.[1] Others have

1. Edmunds, C. W.: The Potency of Digitalis Preparations of the 1936 Pharmacopeia, J. A. M. A. **113**: 284 (July 22) 1939.

differed with him, holding that the increase was greater, up to 50 per cent or more.[2]

Criticisms do not seem to have been offered of the intent of the Revision Committee of the U. S. P. XI to make the digitalis preparations of the U. S. P. conform to international practice. As far as it failed to do exactly what was intended this failure was for technical reasons, centering in part at least around the particular material used as the standard of reference. The Twelfth Revision committee has wisely continued the general policy. Again a reference digitalis powder has been established against which all official digitalis preparations are to be assayed. Although it is this powder that is the legal standard, it has been adjusted to contain the same number of international units per gram as it does U. S. P. XII units. Therefore the tincture of digitalis U. S. P. XII will be of the same potency, for example, as the tincture of digitalis of the British pharmacopeia. This action means that official digitalis preparations will continue to be more potent than those in use during the period prior to 1936.

The U. S. P. XII revision committee has not only adopted a new standard reference powder in place of the old but also has adopted a new method of comparison of digitalis preparations with the official standard. Cats are now to be used for purposes of comparison rather than frogs. Although the communication from the chairman of the Subcommittee on Biological Assays does not discuss the reasons for this change in method it represents apparently recognition of the claims of Gold, Cattell and their associates [3] that assays made on cats give results which more nearly parallel the potency in man than those which use frogs. Change from frogs to cats does not, however, involve acceptance of the cat unit. Indeed, the studies of the Cornell group [4] have shown that it may require 25 cat units of digitalis leaf to digitalize a subject, whereas 3 cat units of "digitaline nativelle" will accomplish the same end. Such studies, of course, have destroyed the value of the "cat unit" as a guide to dosage.

With adoption of both a new reference standard and a new method of assay, confidence of the medical profession in the uniform potency of official preparations should be increased. However, the communication from the chairman of the Subcommittee on Biological Assays properly emphasizes that confusion will continue unless the profession clearly recognizes that there has been an increase in potency of about 25 per cent over preparations in use prior to 1936.

2. Bland, E. F. and White, Paul D.: The Strength of Digitalis in Clinical Use: A Warning, J. A. M. A. **117**: 1243 (Oct. 11) 1941.
3. Gold, Harry; Cattell, McKeen; Kwit, N. T., and Kramer, Morton: J. Pharmacol. & Exper. Therap. **73**: 212 (Oct.) 1941.
4. Gold, Harry; Kwit, N. T., and Cattell, McKeen: J. Pharmacol. & Exper. Therap. **69**: 177 (July) 1940.

A Call To Service

DR. FRANK H. LAHEY
President of the American Medical Association

The establishment by the government of a Procurement and Assignment Agency properly places the responsibility for obtaining medical personnel in the hands of the medical profession. The success of this agency depends entirely on a few basic features: the complete cooperation of medicine in what even the most doubting must now admit is a truly national emergency; an unqualified willingness to serve the country however, wherever and whenever required; and a firm purpose to establish the fact that medicine intends to maintain its place in the forefront as it always has when a patriotic example is of such significance.

AIR RAID HYPOIMMUNITY

Hygienists will find material for thought in Pfannenstiel's [1] recent report of the effect of wartime conditions on the general level of immunity among people of western Europe. For a decade Pfannenstiel and his co-workers have studied the variation in the bacteriostatic and bactericidal power of the blood of animals under variable nutritional and environmental conditions. While they did not assume that variations in serum titer are complete measures of changes of immunity in experimental animals, they did assume that humoral variations are the best available indexes in such studies. The technic adopted for such studies was a modification of the technic endorsed by Wright and Holsen,[2] a constant volume of defibrinated blood mixed with an arbitrary volume of bacterial suspension. The mixture is incubated for twenty-four hours with a control utilizing the same bacterial suspension in melted agar. Comparison of the number of colonies which develop in the two tests gives a ratio which they designated as the "bactericidal index." Thus if 1,000 colonies per unit volume develop in the control agar mixture, while but 200 viable organisms are demonstrable in the incubated blood sample, the "index" (or ratio) is 1,000:200, or 5. An index of 1 thus means a complete absence of bactericidal action. Applying this technic, Pfannenstiel found that the bactericidal index of a rabbit's blood, for example, is often raised thirtyfold (average, from 2.2 to 56.7) within four hours after intravenous injection of neoarsphenamine, with an occasional rabbit showing an increase of as much as one hundred and fifty fold.[3]

1. Pfannenstiel, W., and Dötzer, W.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **99**: 86 (Dec. 4) 1940.
2. Holsen, T.: Ztschr. f. Vitaminforsch. **1**: 3, 1932.
3. Pfannenstiel, W., and Quante, W.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **88**: 1 (May 30) 1936.

In his earlier studies Pfannenstiel used the typhoid bacillus as his test micro-organism.[4] He found, for example, that a diet of turnips increases the bactericidal titer of rabbits and that three days' inanition had an even more beneficial effect. The latter observation is contrary to previous data reported by Meltzer and Norris,[5] who found that the bactericidal action of dog serum was static, "no matter whether it was taken from a well fed, overfed or starving animal." Their studies agree, however, in denying a reduction in humoral immunity as a result of moderate starvation. Pfannenstiel afterward tested the effect of quantitative variations in the daily intake of individual food elements. He found, for example, that a moderate decrease in the daily intake of a mixture of the four vitamins A, B, C and D was without deleterious effects. Subnormal blood titers produced by any cause, however, were increased to normal by the administration of relatively large doses of vitamin C. He concluded from these vitamin tests that in rabbits the bactericidal titer and the vitamin C content of the blood stream are interdependent variables, i. e. they vary parallel with each other.

The immunologic effects of low nutritional level and low vitamin intake became of major importance with the outbreak of the present European war. Bimonthly titrations of bactericidal power and vitamin C content of human blood were therefore begun early in 1940 in the Hygienic Institute, Marburg, Germany. During the first six months the vitamin C content of the blood of selected hospital personnel varied from 2.54 to 14.4 mg. per hundred cubic centimeters. The bactericidal titers of the same individuals varied from 1.00 to 5.04, staphylococci being used as the test micro-organisms. The most obvious fact was the complete lack of parallelism between the vitamin C content and the bactericidal index. The highest recorded bactericidal titer (5.04) was associated with a relatively low vitamin content (5.24 mg.), while the highest vitamin titer (11.46 mg.) was accompanied by low humoral immunity (1.36). The conclusion drawn from these data is that the bactericidal power and the vitamin C content of human blood are independent variables. Partial vitamin C starvation, therefore, has not apparently had deleterious effects on immunity among persons in central Europe. A low total food intake may even be beneficial in conquered countries, since in rabbits from three to five days' total inanition increases the resistance to microbes.

In order to explain observed fluctuations in individual resistance to bacteria, Pfannenstiel called attention to the repeated nocturnal air raid alarms during July and August, with the resulting overwork and loss of sleep. Following each sleepless night half of the selected hospital personnel showed a lowering of bactericidal titer. In 1 case the previous index (5.04) fell two thirds (i. e. to 1.53) following a nocturnal air raid alarm. In a second person there was a complete loss of previous bactericidal power. It may or may not be a coincidence that the second person developed a staphylococcic infection during August, the period of air raid alarm hypoimmunity. Insomnious depression of bactericidal titer was not noted in those who had an opportunity to sleep during the daytime following the night raid.

Disregarding the evident propaganda motivation of this research, evidence of the lack of parallelism between vitamin C intake and bactericidal immunity (or the relatively small amount of vitamin C necessary to maintain maximum humoral efficiency) is of basic clinical interest. Evidence of an insomnious depression of natural immunity is a belated confirmation of a belief long held by physicians. Whether or not data drawn from selected hospital personnel are applicable also to the regional population was not tested by the Nazi investigators.

4. Pfannenstiel, W.: Ztschr. f. Immuntätsforsch. u. exper. Therap. **56**: 389 1927.
5. Meltzer, S. J., and Norris, C.: J. Exper. Med. **4**: 131, 1899.

Current Comment

COLOSTRUM CUTANEOUS TEST FOR PREGNANCY

Several tests have been proposed for furnishing an early reliable diagnosis of pregnancy. An immunologic test, depending on a local cutaneous reaction, has been especially sought. However, thus far only biologic tests are considered reliable. Recently Falls, Freda and Cohen [1] reported a method of cutaneous testing which is claimed to be 98 per cent accurate in pregnant women and 96 per cent accurate in nonpregnant women. In this test a preparation of colostrum is injected into the skin. A faint reaction or none at all is apparent in pregnant women, while the nonpregnant react with a prominent wheal typical of an allergic response. This reaction appears within one-half hour, according to the authors. They tested 358 known nonpregnant women and 265 pregnant women at the time of their report. The test has already been commercialized by G. H. Sherman, M.D., Inc., and physicians are being circularized with the statement that the test is accurate and dependable in the hands of any physician. In view of lack of evidence supporting the results of the original investigators, this premature exploitation is deplored. Physicians may well maintain a skeptical attitude toward the promoter's advertising claims until the original work of Falls and his associates has been adequately confirmed. In this regard complications seem to have developed, as Weisman and Snyder report, in the October issue of the *American Journal of Obstetrics and Gynecology*, page 738, a wide margin of error in the diagnosis of pregnancy by the use of this test. The well established biologic tests need not yet be abandoned.

1. Falls, F. H.; Freda, V. C., and Cohen, H. H.: A Skin Test for the Diagnosis of Pregnancy, Am. J. Obst. & Gynec. **41**: 431 (March) 1941; read before the fifty-third annual meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Excelsior Springs, Mo., Sept. 26, 1940.

MEDICAL PREPAREDNESS

In this section of The Journal each week will appear official notices by the Committee on Medical Preparedness of the American Medical Association, announcements by the Surgeon Generals of the Army, Navy and Public Health Service, and other governmental agencies dealing with medical preparedness, and such other information and announcements as will be useful to the medical profession.

PROCUREMENT AND ASSIGNMENT SERVICE

Office of Civilian Defense Needs Two Physicians Immediately

The United States Public Health Service requires at once two physicians, aged 45 to 60, for important duties in connection with the Office of Civilian Defense. If found suitable, these physicians will be offered commissions in the Reserve Corps of the United States Public Health Service in the grade of Senior Surgeons with pay and allowances of the grade corresponding to that of Lieutenant Colonel in the Army or Commander in the Navy. As soon as commissioned, they will be ordered to active duty and directed to report to the Chief Medical Officer of the Office of Civilian Defense. Duties in general are to represent the Medical Division of the Office of Civilian Defense in the Sixth and Seventh Defense regions (this corresponds to the Sixth and Seventh Army Corps areas), study the Office of Civilian Defense program, interpret the medical program to the state and local defense organizations, maintain contact with state and local defense organizations, establish and maintain liaison with governmental and other medical and health organizations in that area, also liaison with the district public health service offices and inspect emergency medical field units in local civilian defense organizations. Active duty will be for the duration of the emergency. Applications should be sent directly to the Executive Officer, Procurement and Assignment Service, New Social Security Building, room 5654-ODH, Fourth and C streets, Washington, D. C.

FORTY-TWO MORE NEW NAVY MEDICAL OFFICERS

The U. S. Navy Department, Washington, D. C., has announced the appointments of the following forty-two physicians as assistant surgeons in the medical corps of the navy to rank as lieutenants (junior grade):

ANDERSON, D. F., Mount Vernon, Ill.
BAKER, W. S., Jr., Detroit.
BELL, John P., Annapolis, Md.
BURT, Charles W., Detroit.
CAREY, John E., Kenmore, N. Y.
CLEMENT, Conrad C., Babylon, N. Y.
DASLER, Theodore W., Deer Park, Wis.
DAY, Lawrence C., Grayslake, Ill.
deTARNOWSKY, G. O., Bartlett, Ill.
DOUGHERTY, William J., Washington, D. C.
DUNN, John C., Brooklyn.
EISBERG, Harry B., Staten Island, N. Y.
ENGLE, Paul R., Petoskey, Mich.
FALK, Victor S., Wauwatosa, Wis.
FRANTZ, Winthrop S., Sacramento, Calif.
GARNETT, R. W., Jr., Emory University, Ga.
GULLEDGE, W. H., Wadesboro, N. C.
HUNTER, William L., Emory University, Ga.
JACOBY, Jack M., Detroit.
JARDON, F. J., Philadelphia.
JARRETT, Thirl E., Norfolk, Va.
JOHNSON, Frank G., Omaha.
KULCZYCKI, Edward, Scranton, Pa.
LAWRENCE, Richard, Jr., San Francisco.
LEIGHTON, Herbert T., Boston.
LOWMAN, E. W., San Diego, Calif.
LYONS, Harold A., Brooklyn.
MACKBY, Maxwell J., New York.
MANN, B. F., Jr., Hartford, Conn.
MILLS, Dawson A., Pittsburgh.
MOORE, Jack D., Montclair, N. J.
MURPHY, John M., Portsmouth, Va.
NIFOROPULOS, J. A., Schenectady, N. Y.
ORR, Robert B., Brooklyn.
RICKERD, Charles L., Seattle.
SHAFIROFF, Charles, Jamaica.
SPAULDING, E. M., New Albany, Ind.
STUCK, Jaul L., Jonesboro, Ark.
UMIKER, William O., Buffalo.
WHITE, W. A., Jr., Pearl Harbor, Hawaii.
WHITELY, Ralph D., Washington, D. C.
YOOD, Norman L., New York.

TYPHOID VACCINE OUTPUT INCREASED 800 PER CENT

Typhoid-paratyphoid vaccine is made at the Army Medical School in the largest laboratory of its kind in the world. The planting and harvesting rooms, made of monel metal and glass, are sterilized with live steam. During the various steps of vaccine making the chambers are sealed, and conditioned air sterilized by washing and passing through ultraviolet light is pumped in under positive pressure. All the typhoid-paratyphoid vaccine required for use by the Army, the Navy, the Civilian Conservation Corps and other federal departments is made at this laboratory, which at peak production can provide one and a half million doses of the vaccine every week. During the last fiscal year the output was increased over 800 per cent.

PLAN TO RELIEVE SHORTAGE OF NURSES

To relieve the shortage of nurses in hospitals, clinics, public health and field nursing agencies, the American National Red Cross and the Office of Civilian Defense, Washington, D. C., have jointly undertaken a project to train 100,000 volunteer nurses aides, with whose assistance graduate nurses may extend their services to many more patients. The volunteer aide will work under the supervision of a nurse in performing certain nontechnical tasks in order that graduate nurses may be released for highly technical duties.

The local office of Civilian Defense and the local chief of Emergency Medical Service in communities where the program for training nurses aides is undertaken will assist the Red Cross and the Civilian Defense Volunteer Office in enrolling desirable applicants for training as nurses aides and will assist local chapters to make arrangements with general hospitals to serve as training centers. The first half of the course of instruction, consisting of lectures and demonstrations, will be given in the Red Cross Chapter Hospital or other suitable place, while the second half of the course will be given in a hospital selected by the Red Cross and the Office of Civilian Defense as a training center.

The Red Cross has outlined in its publication "Chapter Organization and Administration of Red Cross Volunteer Nurses Aides Corps" the duties of nurses aides which will be subject to the approval of individual institutions. Among other activities, the nurses aides in the hospitals may take care of personal belongings of patients, make beds, feed helpless patients, clean dressing trays, take patients to and from treatment rooms, help with the admission and discharge of patients, care for ambulatory patients, take care of linen closets and of rubber goods, and in dispensaries and clinics may serve as interpreters in foreign languages, dress and undress children, clean and put away instruments, help weigh and measure, assist in taking physicians' notes and help with inventories.

The Office of Civilian Defense, Washington, D. C., has prepared a syllabus of the courses to be given nurses aides, and the American Red Cross has published a Guide for the Training of Volunteer Nurses' Aides, and copies of these are available from local Red Cross chapters or from Regional Civilian Defense Offices.

Nurses aides are to serve without remuneration. The time for the course of instruction is eighty hours. After the first thirty-four hours of the course the aides will wear the uniform with insignia, described in the Guide for the Training of Volunteer Nurses' Aides. Membership in the Nurses Aides Corps requires the aide to be between 18 and 50 years of age when

enrolled, in satisfactory physical condition, a graduate from high school or equivalent. One hundred and fifty hours of service must be given in each calendar year, preferably in a three months period, and the first one hundred and fifty hours must be spent in hospital wards before assignments to clinics and field nurses' organizations. In time of war or other national emergency, aides must be prepared to serve whenever and for as long a period as needed.

BALANCED MEALS FOR SOLDIERS

A master menu, containing all the necessary nutritional requirements for an active soldier and ample variety and quantity of foods, is under preparation by the Army Quartermaster Corps. It will be divided into twelve units, one for each month, containing submenus for every day in the week. The November series was sent out to all Corps Area Commanders. The menus can be used as prepared or adapted to local needs. On the basis of the master menu, monthly menus will be prepared for use in Army posts, camps and cantonments.

The master menu is prepared with the average soldier in mind. He is of medium height and weight but, being engaged in strenuous activities, is hungrier than the average civilian of the same build. The average number of calories daily per man is about 4,500. The menus call for more minerals than meet the requirements of the Nutritional Committee of the National Research Council: iron in excess of 20 mg., phosphorus 2.2 Gm. and calcium 1.1 Gm. The vitamin A total daily is about 7,000 international units, vitamin C 115 mg., vitamin B_1 (thiamine hydrochloride) 3 mg. and vitamin B_2 (riboflavin) 3.1 mg.

Care is exercised to insure that the soldier shall not get a surplus of starchy foods at any one meal. Peas, for example, are not served with corn or beans, nor potatoes with macaroni or spaghetti. Where canned fruits and vegetables are listed in the menus, fresh fruit and vegetables should be substituted if they are available, according to the locality. The following is a typical menu from the master menu for a November day:

Breakfast	Dinner	Supper
Orange	Barley soup	Spareribs
Assorted cereal	Soft roast beef	Boiled potatoes
Fresh milk	Creamed potatoes	Buttered cabbage
Cheese omelet	Spinach	Cinnamon buns
Lyonnaise potatoes	Pickled beet salad	Bread and butter
Bread and butter	Bread and butter	Coffee
Coffee	Coffee	

REGIONAL MEDICAL OFFICERS FOR CIVILIAN DEFENSE

The Office of Civilian Defense, Washington, D. C., has organized on a regional basis, with regions corresponding to the U. S. Army Corps Areas. A regional director has been appointed for each region with headquarters in the same city in which the corps area headquarters are located. Regional medical officers have been appointed as follows:

First Civilian Defense Region: Dr. Allan M. Butler, 101 Milk Street, Boston.
Second: Dr. H. Van Zile Hyde, 111 Eighth Avenue, New York.
Third: Dr. W. Ross Cameron, 400 Cathedral Street, Baltimore.
Fifth: Dr. William S. Keller, 425 Cleveland Avenue, Columbus, Ohio.
Eighth: Dr. Witten B. Russ, Room 1014, Majestic Building, San Antonio, Texas.
Ninth: Dr. Wallace Hunt, 233 Sansome Street, San Francisco.

These physicians have been commissioned as senior surgeons in the U. S. Public Health Service.

HISTORY OF EXPANSION OF MEDICAL ACTIVITIES DURING THE PRESENT EMERGENCY

A history of the expansion of medical activities in the United States during the present emergency is now being compiled under the direction of Dr. John F. Fulton, chairman of the subcommittee on historical records of the Committee on Information, Division of Medical Sciences of the National Research Council. The history will analyze the expanded activities of the civilian and governmental medical agencies from April 1939, when defense expansion began, until the end of the emergency.

According to the *Army and Navy Journal*, November 22, the general history of medical activities during the emergency will be contained in nine volumes, a number subject to change.

Major Gen. James C. Magee, Surgeon General of the Army, has stated that special emphasis will be placed on the history of the Medical Department divisions since April 1939, when an authorized increase in the Air Corps and the forces at Panama signaled the beginning of defense expansion. General Magee has requested Col. Albert G. Love, M. C., retired, to return to active duty to supervise the present medical department history. Colonel Love is organizing the history of the Medical Department since April 1939, which tentatively will be contained in seven volumes, supplementing the nine general volumes.

Colonel Love, who is said to be an authority on analytic histories of the Medical Department, and Capt. Lewis H. Roddis, M. C., U. S. Navy, are co-editors of volume VI of the general nine volume history, which will contain the "Medical History of Individual Operations and Engagements." Col. George R. Callender, M. C., is editor of volume II, which will give the history of "Offensive Weapons in Relation to Injury." The only other histories of Medical Department activities are those which are concerned with the Civil War and World War I. The present history, when completed, will make the third analytic work completed and the first to contain an analysis of all medical activities—civilian, military and governmental.

PROTECTIVE PROCEDURES FOR HOSPITALS

Dr. George Baehr, chief medical officer of the Office of Civilian Defense, Washington, D. C., has appointed the following subcommittee of the Advisory Board of the Medical Division, Office of Civilian Defense, to prepare recommendations on protective procedures for hospitals in the event of belligerent action:

Dr. Robin C. Buerki, director of hospitals of the University of Pennsylvania, Philadelphia, chairman.
Dr. Willard C. Rappleye, commissioner of hospitals, New York.
Dr. Asahel J. Hockett, superintendent of Touro Infirmary, New Orleans.
Dr. Anthony J. J. Rourke, medical superintendent of Stanford University Hospitals, San Francisco.
Dr. Joseph Turner, director of Mount Sinai Hospital, New York.
Dr. Huntington Williams, commissioner of health of Baltimore.

The subcommittee held its first meeting in New York on November 8. With Dr. James M. Mackintosh, former chief medical officer of the Scottish Ministry of Health, as a guest to advise the group, measures for preventing or minimizing damage to buildings, handling of casualties, evacuation, provision and protection of supplies, and training of personnel for specific duties in case of bombing were discussed. The basis for discussion was a study made by a committee of the American Hospital Association on physical defense of hospitals. It is expected that a report will be issued jointly by the hospital association committee and the committee representing the Office of Civilian Defense.

The basic organization for rescue work in a given area consists of three essential groups with a central control, police, rescue and fire services, air raid precaution control and medical service.

HUGE REFRIGERATORS FOR ARMY FOOD SUPPLIES

Some of the mammoth army ice boxes will hold enough perishable food—meat, butter, eggs, fruit and vegetables—to feed a division of about fifteen thousand soldiers for two weeks. Recently a quartermaster statistician observed in one of these ice boxes 50 tons of meat, 6 tons of butter, 8,000 dozen eggs and, among other items, 25 tons of fresh fruits and vegetables.

HEALTH EDUCATION CONSULTANTS TO BE ASSIGNED TO DEFENSE AREAS

The rapid growth in industrial and governmental production has caused unusual concentrations of population and increasing health problems. To assist state, county and local health officials in coping with these problems, the U. S. Public Health Service is planning to appoint health education consultants to various defense areas. The positions, paying $2,600 to $3,800 a year, will be filled through the open competitive examination process and the Federal Civil Service Commission has just issued the

examination announcement. A written test will not be given, but applicants will be rated on their qualifications as shown in their applications and corroborative evidence.

Appointees will work with local health officers and their staffs, advising them as to methods and procedures of health education, such as individual instruction through interview, group instruction through discussions, talks, lectures and other educational technics. To qualify for the positions, applicants must have completed a four year college course, including or supplemented by special study—or, for the assistant grade, experience—in public health. In addition they must have had experience in public health education work coordinating the activities of all organized health groups in a community for the purpose of promoting a public health program. This experience must have been in a federal, state or official local public health department or in a voluntary agency such as the Red Cross or the Tuberculosis Association.

Applications must be filed with the Civil Service Commission in Washington, D. C., not later than December 11. The examination announcement giving detailed requirements can be obtained at any first or second class post office or at the central office in Washington, D. C.

ARMY RESERVE OFFICERS ORDERED TO ACTIVE DUTY

WAR DEPARTMENT

The following additional medical reserve officers have been ordered to extended active duty by the War Department, Washington, D. C.:

ARGO, William Lind, 1st Lieut., Oakland, Calif.
BULLINGTON, Bert Montell, 1st Lieut., Ann Arbor, Mich.
FULTZ, George Simeon, Jr., 1st Lieut., Butterworth, Va.
REICHEL, John, Jr., 1st Lieut., Wynnewood, Pa.
ROSEN, Victor Hugo, 1st Lieut., Baltimore.
SIEBECKER, Karl LaFollette, 1st Lieut., Los Angeles.
THOMPSON, Robert Philp, 1st Lieut., San Francisco.
WALKER, Douglass Willey, 1st Lieut., New Haven, Conn.
WEINSTEIN, Harry, 1st Lieut., Chowchilla, Calif.
WILLERS, Carl Elmer, 1st Lieut., San Francisco.

Orders Revoked

BERMAN, Lawrence, 1st Lieut., Detroit.
JOHNSON, Carroll Allen, Jr., 1st Lieut., Healdton, Okla.
MICKEL, Arthur A., 1st Lieut., Benicia, Calif.
RYAN, Bernard F., 1st Lieut., Ahwahnee, Calif.
SCHILDHAUS, Andrew I. E., 1st Lieut., Burlington, Vt.

CORRECTION

War Department.—The following names of medical reserve officers were erroneously published as "relieved from active duty" in THE JOURNAL, September 6, under the Ninth Corps Area. These names should have been published under "War Department" as "ordered to active duty": Major Albert Guernsey Clark, San Francisco, and Lieuts. Samuel S. Blankstein, Milwaukee; Irving Lawrence Greenberg, Atlanta, Ga.; John Joseph Manning, Chicago; John Edward Naugle, Denver; Ernest Boris Newman, Brooklyn; Phosa David Nutter, Russellville, W. Va.; Samuel Sidney Platt, Chicago; Reno Russell Porter, Brookline, Mass.; Peter Albert Reierson, Chicago; Owen Bernard Royce, Oklahoma City; Thomas Robert White, Redlands, Calif., and William Luther White, Philadelphia.

FIRST CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, First Corps Area, which comprises the states of Maine, Vermont, New Hampshire, Rhode Island, Massachusetts and Connecticut:

CHAIT, Sidney A., 1st Lieut., Middletown, Conn., Fort Banks, Mass.
DEL CAMPO, Dante, 1st Lieut., Lynn, Mass., Westover Field, Mass.
CORRIGAN, Francis V., 1st Lieut., Providence, R. I., Windsor Locks, Conn.
CRISCUOLO, Joseph A., Jr., 1st Lieut., New Haven, Conn., Fort H. G. Wright, N. Y.
HARRIS, Oliver J., 1st Lieut., Boston, Westover Field, Mass.
LAMB, Francis D., 1st Lieut., West Warwick, R. I., Windsor Locks, Conn.
MORRISON, Jonathan I., 1st Lieut., Waban, Mass., Westover Field, Mass.
PATTERSON, John C., 1st Lieut., Boston, Manchester, N. H.
SIMON, Norman, 1st Lieut., Lawrence, Mass., Westover Field, Mass.
WALKER, Donald A., 1st Lieut., Worcester, Mass., Army Air Base, Bangor, Maine.
YAVAROW, Mully Millin, 1st Lieut., Everett, Mass., Manchester, N. H.

Orders Revoked

BRIER, Hyman D., 1st Lieut., Bridgeport, Conn.
DOERNER, Alexander A., 1st Lieut., Chelsea, Mass.
FELDERMAN, Jacob, 1st Lieut., Rutland Heights, Mass.
PEARSON, Grosvenor B., 1st Lieut., Foxboro, Mass.
SODA, William E., 1st Lieut., Bridgeport, Conn.

SECOND CORPS AREA

The following additional medical reserve corps officers have been ordered to active duty by the Commanding General, Second Corps Area, which comprises the states of New York, New Jersey and Delaware:

ANDERSON, Orrin E., 1st Lieut., Clifton Springs, N. Y., Fort Du Pont, Del.
BERGER, Harold R., 1st Lieut., Elizabeth, N. J., Fort Jay, N. Y.
BLUESTONE, Leon, 1st Lieut., Brooklyn, Camp Claiborne, La.
HELDMAN, Arthur, 1st Lieut., New York, Camp Shelby, Miss.
HOOD, George B., 1st Lieut., Camden, N. J., Fort Bragg, N. C.
LAPIDUS, Bernard, 1st Lieut., New York, Pine Camp, N. Y.
MARRELLA, Louis F., 1st Lieut., Jersey City, N. J., Camp David, N. C.
MOUNTAIN, Edward R., 1st Lieut., Olean, N. Y., Fort Ontario, N. Y.
PISANI, Antonio J., 1st Lieut., New York, Pine Camp, N. Y.
SMITH, Henry T., Major, New York, Camp Wheeler, Ga.
TURNER, Fennell P., 1st Lieut., New York, Camp Claiborne, La.
VON SALZEN, Charles F., 1st Lieut., Kings Park, L. I., N. Y., Camp Upton, N. Y.

Orders Revoked

VALENTE, Michael A., 1st Lieut., Fort Benning, Ga.

THIRD CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, Third Corps Area, which comprises the states of Pennsylvania, Virginia, District of Columbia and Maryland:

DEAN, James Seay, Captain, Pennhurst, Pa., Fort Eustis, Va.
GRAU, John Karl, 1st Lieut., Beaver Falls, Pa., Fort George G. Meade, Md.
HAMPSEY, Joseph William, Captain, Pitsburgh, Camp Lee, Va.
HENDERSON, Charles Henry, Captain, Norton, Va., Fort Belvoir, Va.
MASLAND, Richard Lambert, 1st Lieut., Philadelphia, Fort George G. Meade, Md.
MICELI, Joseph, 1st Lieut., Baltimore, Fort Belvoir, Va.
OPPLEMAN, Herman Frank, 1st Lieut., Richmond, Va., MacDill Field, Fla.
PROCOPIO, James Joseph, 1st Lieut., Shamokin, Pa., Camp Claiborne, La.
RALSTON, Edgar Lee, 1st Lieut., Murrysville, Pa., Fort George G. Meade, Md.
RANDALL, Robert Stewart, 1st Lieut., Washington, D. C., Fort Dix, N. J.
SAMPLE, Hyde Glenn, Jr., 1st Lieut., Pittsburgh, Edgewood Arsenal, Md.
SCHORNICK, James Curtis, 1st Lieut., Philadelphia, Fort George G. Meade, Md.
SETTLE, William Booth, Captain, Baltimore, Indiantown Gap Military Reservation, Pa.

SINDACO, Mario Secondo, 1st Lieut., Plains, Pa., Indiantown Gap Military Reservation, Pa.
SPIEGEL, Herbert X., 1st Lieut., Washington, D. C., Fort George G. Meade, Md.

Orders Revoked

BAILEY, Charles P., 1st Lieut., Philadelphia.
BARNETT, Luke J., 1st Lieut., Pittsburgh.
BASTIEN, Henry L., Captain, Arlington, Va.
CAMPBELL, Hawes, Jr., 1st Lieut., Gary, W. Va.
CHESNUT, John L., 1st Lieut., Mountain Grove, Va.
COOPER, George, Jr., 1st Lieut., Charlottesville, Va.
COVALESKY, Victor J., 1st Lieut., Aspinwall, Pa.
CRAVOTTA, Charles A., Captain, Pittsburgh.
ERHARD, Gerald A., 1st Lieut., Curwensville, Pa.
FRONCZEK, William M., 1st Lieut., Pittsburgh.
GATES, Earle C., Captain, Chesterfield, Va.
HURST, John W., 1st Lieut., Altoona, Pa.
ITSCOITZ, Seymour E., 1st Lieut., McKeesport, Pa.
KAUFMAN, Benjamin V., 1st Lieut., Taylor, Pa.
LIM, John P., Captain, Stroudsburg, Pa.
LYON, William C., 1st Lieut., Ardmore, Pa.
MENSH, Maurice, 1st Lieut., Washington, D. C.
MESSER, William A., Captain, Pittsburgh.
MITCHELL, John A., 1st Lieut., Monaca, Pa.
PUGH, George E., 1st Lieut., Scranton, Pa.
RATTENNE, Edward, 1st Lieut., Elkton, Va.
RUEHL, William W., 1st Lieut., Pittsburgh.
SCHEIN, Robert A., 1st Lieut., Pittsburgh.
SCHWARTZMAN, Joel J., 1st Lieut., Oaklyn, N. J.
SHENKIN, Henry A., 1st Lieut., Philadelphia.
SNOW, Lee B., 1st Lieut., Washington, D. C.
THOMAS, Preston W., 1st Lieut., Mayview, Pa.
VORBRINCK, Thomas M., Captain, Norfolk, Va.
WALMER, Charles R., 1st Lieut., Pittsburgh.
WILCOX, Homer B., Jr., 1st Lieut., Kingston, Pa.
WILSON, John M., Jr., 1st Lieut., Pittsburgh.
WITKIN, Leon A., 1st Lieut., Philadelphia.

FOURTH CORPS AREA

The following additional medical reserve corps officers have been ordered to active duty by the Commanding General, Fourth Corps Area, which comprises the states of Tennessee, North Carolina, South Carolina, Alabama, Georgia, Mississippi, Florida and Louisiana.

BOLGLA, Julius Hill, 1st Lieut., Spartanburg, S. C., Fort Barrancas, Fla.
COHEN, Nace Ralph, 1st Lieut., Montgomery, Ala., Camp Gordon, Ga.
DeFREESE, Samuel J., 1st Lieut., Atlanta, Ga., Fort Barrancas, Fla.
DITATA, Domenick, Captain, Montgomery, Ala., Fort Barrancas, Fla.
GAILLARD, Peter C., Jr., 1st Lieut., Beaufort, S. C., Fort McClellan, Ala.
ROZIER, John S., 1st Lieut., Leesville, La., Camp Forrest, Tenn.
SMITH, Charles G., III, 1st Lieut., Rocky Mount, N. C., Fort Barrancas, Fla.

Orders Revoked

EVERS, Herbert Ray, 1st Lieut., Andalusia, Ala.
MOLE, John William, Captain, Brunson, S. C.
ROSS, Sam H., 1st Lieut., Seneca, S. C.
SAMPOGNARO, Vincent J., 1st Lieut., Monroe, La.

FIFTH CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, Fifth Corps Area, which comprises the states of Ohio, West Virginia, Indiana and Kentucky:

ABRAHAMSON, Ira Arthur, Lieut. Colonel, Cincinnati, Camp Davis, N. C.
ADELMAN, Jack Arnold, 1st Lieut., Columbus, Ohio, Drew Field, Fla.
ADLER, Raymond Nicholas, 1st Lieut., Evansville, Ind., Fort McClellan, Ala.
AKE, Loren Francis, 1st Lieut., Cambridge City, Ind., Camp Polk, La.
ALBERTSON, Frank P., 1st Lieut., Trafalgar, Ind., Camp Shelby, Miss.
ALEXANDER, Eugene James, 1st Lieut., Evansville, Ind., Camp Polk, La.
ARTHUR, Robert Daniel, Captain, Springfield, Ohio, Camp Davis, N. C.
ASH, Edwin Everett, 1st Lieut., Forest, Ohio, Luke Field, Ariz.
AVERY, Bruce Franklin, 1st Lieut., Whiting, Ind., Camp Grant, Ill.
BAKER, Leslie Mayer, 1st Lieut., Aurora, Ind., Fort Oglethorpe, Ga.
BARKER, Harold Jerome, 1st Lieut., Cleveland Heights, Ohio, Camp Shelby, Miss.
BARNARD, William Harvey, Captain, Elizabethtown, Ky., Camp Wheeler, Ga.
BELINKY, Nathan Donald, 1st Lieut., Youngstown, Ohio, Fort Santiago, Philippine Islands.
BENEDETTO, Eugene Rudolf, 1st Lieut., Alliance, Ohio, Fort Bragg, N. C.
BENNETT, Jene Richard, 1st Lieut., Plymouth, Ind., Air Base, Lowry Field, Colo.
BEREN, Irvin Bernard, 1st Lieut., Cincinnati, Fort Bragg, N. C.
BERGER, Irving Lester, 1st Lieut., Cleveland Heights, Ohio, Fort Jackson, S. C.
BERGER, Peary Benn, Captain, Englewood, Ohio, Camp Shelby, Miss.
BERSHADSKY, Solomon, 1st Lieut., Cincinnati, Air Base, Gunter Field, Ala.
BERTLING, Marion Henry, 1st Lieut., McComas, W. Va., Fort Bragg, N. C.
BINZER, Isadore Irving, 1st Lieut., Toledo, Ohio, Fort Knox, Ky.
BOICE, Robert Roland, Captain, Pomeroy, Ohio, Camp Blanding, Fla.
BORER, Raymond John, Captain, Toledo, Ohio, Fort Jackson, S. C.
BOSWORTH, Nathaniel Lewis, Jr., 1st Lieut., Lexington, Ky., Fort Knox, Ky.
BOWERS, Copeland Connelly, Captain, Kokomo, Ind., Sheppard Field, Texas.
BOWERS, Garvey Bruce, Captain, Kimball, W. Va., Losey Field, Puerto Rico.
BRADFORD, Bert, Jr., 1st Lieut., Charleston, W. Va., Camp Forrest, Tenn.
BRAMMER, Fred Emerson, Major, Huntington, W. Va., Camp Wheeler, Ga.
BREMEN, Harry A., 1st Lieut., Dayton, Ohio, Ellington Field, Texas.
BROOKIE, Roger William, Major, Flora, Ind., Camp Croft, S. C.
BROWN, Robert Henry, 1st Lieut., Cincinnati, Camp Livingston, La.
BRUMBAUGH, Joseph John, Major, Canton, Ohio, Station Hospital, San Juan, Puerto Rico.
BRUMLEY, Donald Richard, Captain, Tiffin, Ohio, Columbus General Depot, Columbus, Ohio.
BUNIN, Simon S., 1st Lieut., Cleveland, Camp Shelby, Miss.
BURGIN, Bernard, 1st Lieut., Cincinnati, Camp Shelby, Miss.
BURNIKEL, Raymond Henry, 1st Lieut., Evansville, Ind., Lowry Field, Colo.
BUSH, Joe Milbert, Captain, Mount Sterling, Ky., Fort Benning, Ga.
BUTTE, Clarence Ivanhoe, Jr., 1st Lieut., Matoaka, W. Va., Camp Claiborne, La.
CAFARO, Secondo Raymond, 1st Lieut., Youngstown, Ohio, Camp Blanding, Fla.
CALO, Frank James, 1st Lieut., Cleveland, Camp Shelby, Miss.
CAMPBELL, George Warren, 1st Lieut., Columbus, Ohio, Camp Davis, N. C.
CAMPBELL, Leland Eston, 1st Lieut., Cleveland, Ellington Field, Texas.
CAMPION, Woodrow Marcellus, 1st Lieut., Cleveland, Fort Bragg, N. C.
CARNEY, Wilfred Ignatius, 1st Lieut., Youngstown, Ohio, Camp Wheeler, Ga.
CHATTIN, Herbert Odell, 1st Lieut., Vincennes, Ind., Brookley Field, Ala.
CLOUSE, Paul Alexander, 1st Lieut., Evansville, Ind., Kelly Field, Texas.
COHEN, Arthur Robert, 1st Lieut., Springfield, Ohio, 2d Army Depot, Gurdon, Ark.
COHEN, Benjamin Bainis, 1st Lieut., East Chicago, Ind., Fort Jackson, S. C.
COHEN, Morris, 1st Lieut., Louisville, Ky., Camp Polk, La.
COLLINS, James Robert, 1st Lieut., Xenia, Ohio, Fort Bragg, N. C.
COOK, Robert Gibson, 1st Lieut., Toledo, Ohio, Camp Forrest, Tenn.
CRANK, Gilbert O., Major, Lawton, W. Va., Camp Davis, N. C.
CREAMER, David Myers, 1st Lieut., Bellaire, Ohio, Fort Jackson, S. C.
DAVIS, Alexander Nick, 1st Lieut., Dayton, Ohio, Camp Shelby, Miss.
DAVIS, William, 1st Lieut., Crestline, Ohio, Fort Dix, N. J.
DeARMOND, Albert Murray, Major, Indianapolis, Carlisle Barracks, Pa.
DOAK, Alfred Deaderick, 1st Lieut., Shelbyville, Ky., Randolph Field, Texas.
DOBKIN, Arthur, Captain, Akron, Ohio, Borinquen Field, Puerto Rico.
DOUGHERTY, John Edwin, 1st Lieut., Canton, Ohio, Randolph Field, Texas.
DUTROW, Howard Victor, Major, Dayton, Ohio, Borinquen Field, Puerto Rico.
EBERLE, William Harrison, 1st Lieut., Ashtabula, Ohio, Fort Jackson, S. C.
EDEL, Charles Henry, Major, Ashtabula, Ohio, Borinquen Field, Puerto Rico.
EDELSTEIN, Joseph Bernard, Captain, Toledo, Ohio, Camp Shelby, Miss.
EGOLF, Charles Foster, Jr., 1st Lieut., Cincinnati, Fort Bragg, N. C.
EPSTEIN, Samuel, 1st Lieut., Struthers, Ohio, Fort Jackson, S. C.
FARGO, Warren Conrad, Lieut. Colonel, Cleveland, Fort Sam Houston, Texas.
FASOLDT, Laurence Otto, 1st Lieut., Glendale, Ohio, Randolph Field, Texas.
FINK, Joseph John, Captain, University Heights, Ohio, Camp Shelby, Miss.
FISH, John Spencer, 1st Lieut., Cleveland Heights, Ohio, Fort Bragg, N. C.

FLAX, Ellis, 1st Lieut., Cincinnati, Camp Shelby, Miss.
FLEETWOOD, Raymond Anson, Captain, Nappanee, Ind., Camp Lee, Va.
FLORA, Joseph Oren, 1st Lieut., Indianapolis, Fort Benjamin Harrison, Ind.
FLORIDIS, Gregory George, 1st Lieut., Dayton, Ohio, Fort Bragg, N. C.
FORWARD, Donald De Klyn, 1st Lieut., Ashtabula, Ohio, Camp Shelby, Miss.
FRANCIS, Carl C., 1st Lieut., Cleveland, Camp Forrest, Tenn.
FREDERICK, Victor Ray, 1st Lieut., South Charleston, Ohio, Camp Davis, N. C.
GAILLARD, Ernest, Jr., 1st Lieut., Louisville, Ky., Randolph Field, Texas.
GARRED, Emery William, 1st Lieut., Louisa, Ky., Fort McClellan, Ala.
GATES, Edmund Oliver, Captain, Welch, W. Va., Camp Blanding, Fla.
GIBER, Philip Bernard, 1st Lieut., Girard, Ohio, Camp Davis, N. C.
GINN, Guy Agustus, Captain, Springfield, Ohio, Fort Bragg, N. C.
GLACKMAN, John Clay, Lieut. Colonel, Rockport, Ind., Fitzsimons General Hospital, Denver.
GLIER, James Randall, 1st Lieut., Greenfield, Ohio, Camp Shelby, Miss.
GOLDBERG, Samuel David, 1st Lieut., Youngstown, Ohio, Camp Davis, N. C.
GOLDSTEIN, Joseph Leon, 1st Lieut., Louisville, Ky., Fort Benning, Ga.
GOLZ, Harold Habich, 1st Lieut., Clarksburg, W. Va., Fort Oglethorpe, Ga.
GOODMAN, Hubert Thorman, 1st Lieut., Terre Haute, Ind., Fort Oglethorpe, Ga.
GOODMAN, Lawrence Hayse, Captain, Findlay, Ohio, Fort Jackson, S. C.
GOULD, Joseph Irwin, 1st Lieut., Toledo, Ohio, Fort Jackson, S. C.
GRAY, James Howard, Captain, Clendenin, W. Va., Camp Davis, N. C.
GREENBERG, Marcel, 1st Lieut., Cleveland, Fort Jackson, S. C.
GREGG, Walter Kimpton, Captain, Dayton, Ohio, Camp Lee, Va.
GRIFFIN, Robert James, 1st Lieut., Lexington, Ky., Fort McClellan, Ala.
GRISELL, Ted Lewis, 1st Lieut., Mitchell, Ind., Camp Shelby, Miss.
GROSS, Theodore Anthony, 1st Lieut., Cleveland, Fort Jackson, S. C.
GROVE, Robert Houston, 1st Lieut., Scircleville, Ind., Camp Shelby, Miss.
HAGGARD, Gordon Hill, Captain, Indianapolis, Fort Oglethorpe, Ga.
HAMSHER, John Fremont, Major, St. Paris, Ohio, Camp Davis, N. C.
HANTMAN, Meyer, Captain, Cleveland, Fort Jackson, S. C.
HARMAN, Howard Engler, Lieut. Colonel, Chillicothe, Ohio, Camp Davis, N. C.
HARRISON, Francis Regis, 1st Lieut., East Liverpool, Ohio, Fort Bragg, N. C.
HAZEL, Woodrow Sidney, 1st Lieut., Youngstown, Ohio, Lowry Field, Colo.
HENGEN, Henry Edward, 1st Lieut., Amherst, Ohio, Fort Jackson, S. C.
HICKMAN, Warren R., 1st Lieut., Logansport, Ind., Fort Thomas, Ky.
HOLMES, Edward Buchanon, Captain, Parkersburg, W. Va., Fort Knox, Ky.
HOLMES, Will Wanee, Major, Logansport, Ind., Camp Croft, S. C.
HORGER, William Jennings, 1st Lieut., East Liverpool, Ohio, Camp Shelby, Miss.
HULLINGER, Clarence William, Major, Springfield, Ohio, Camp Wheeler, Ga.
HUMPERT, Joseph Herman, 1st Lieut., Erlanger, Ky., Camp Wheeler, Ga.
HUMPHRIES, John Kern, 1st Lieut., Belle Center, Ohio, Camp Shelby, Miss.
HUNTER, Howard Marston, 1st Lieut., Hamilton, Ohio, Camp Shelby, Miss.
HUNTER, William Byrd, Major, Huntington, W. Va., Fitzsimons General Hospital, Denver.
IPP, Herman Harry, 1st Lieut., Youngstown, Ohio, Kelly Field, Texas.
JACOB, Samuel Sprigg, III, Captain, Fairmont, W. Va., Fort McClellan, Ala.
JAUCH, Roland Schuster, Captain, Rocky River, Ohio, Fort Sam Houston Texas.
JOHN, Henry Jerry, Major, Cleveland, Camp Wheeler, Ga.
JOHNSON, Robert Ben, 1st Lieut., Butlerville, Ind., Fort Thomas, Ky.
JOHNSTON, Coleman Carnegie, 1st Lieut., Lexington, Ky., Fort Knox, Ky.
JOHNSTON, Walter Bailey, Major, Cleveland, Fort Jackson, S. C.
JONES, Paul Herdman, Captain, Stockdale, Ohio, Camp Davis, N. C.
JUNKERMAN, Carl Schulze, Major, Columbus, Ohio, Camp Shelby, Miss.
JUNKERMANN, Edgar Burnett, Major, Columbus, Ohio, Fort Custer, Mich.
KALMON, Morris M., Captain, Akron, Ohio, Fort Buchanan, Puerto Rico.
KANE, Maurice M., Captain, Greenville, Ohio, Fort Jackson, S. C.
KARLAN, A. Morton, 1st Lieut., Springfield, Ohio, Fort Sill, Okla.
KAUFFMAN, Sidney Albert, 1st Lieut., Indianapolis, Fort Jackson, S. C.
KELLY, Fred Robert, 1st Lieut., Cleveland, Fort Jackson, S. C.
KISH, Louis Stephen, 1st Lieut., Cleveland, Camp Shelby, Miss.
KLOPFER, Edward George, 1st Lieut., Columbus, Ohio, Camp Shelby, Miss.
KNISELY, Alan DeGrief, Major, Lima, Ohio, Camp Wheeler, Ga.
KNISELY, Earl Perry, 1st Lieut., Columbus, Ohio, Camp Claiborne, La.
KRAUSE, George Robert, Captain, Cleveland Heights, Ohio, Fort Jackson, S. C.
KRESS, George Lester, Captain, Warsaw, Ind., Camp Grant, Ill.
KUNTZ, Clarence Ignatius, Captain, Fremont, Ohio, Camp Blanding, Fla.
LACOCK, Wilford Clare, 1st Lieut., Beaverdam, Ohio, Fort Bragg, N. C.
LAPP, Henry Thomas, 1st Lieut., Wilmington, Ohio, Camp Forrest, Tenn.
LAZARUS, Lawrence B., 1st Lieut., Cleveland, Camp Forrest, Tenn.
LEDERER, Henry David, 1st Lieut., Cincinnati, Fitzsimons General Hospital, Denver.
LEUCHTAG, Harry Herman, 1st Lieut., Akron, Ohio, Camp Forrest, Tenn.
LOEHR, William McWhorter, 1st Lieut., Indianapolis, Fort Sam Houston, Texas.
LOTZOFF, Harold Alvin, 1st Lieut., Lima, Ohio, Fort Jackson, S. C.
LUCAS, Marvin Andrew, 1st Lieut., Louisville, Ky., Camp Lee, Va.
MANNINO, Rosario Gilbert, 1st Lieut., Newark, Ohio, Camp Lee, Va.
MARSHALL, John Hugh, Major, Findlay, Ohio, Letterman General Hospital, San Francisco.
MARTIN, George Isaac, 1st Lieut., Blanchester, Ohio, Camp Forrest, Tenn.
MASON, Everett Elmore, 1st Lieut., Evansville, Ind., Camp Blanding, Fla.
MATTEO, Michael Louis, 1st Lieut., Wickliffe, Ohio, Fort Jackson, S. C.
MARYANSKI, William Howard, 1st Lieut., Akron, Ohio, Camp Forrest, Tenn.
McALLISTER, Thurman Francis, 1st Lieut., Coshocton, Ohio, Fort Bragg, N. C.
McCALL, Edward Warner, 1st Lieut., Portsmouth, Ohio, Fort Bragg, N. C.
McKINLEY, Melvin Paul, Captain, East Cleveland, Ohio, New Orleans Air Base, La.
McMAHAN, Virgil Carrol, 1st Lieut., Bedford, Ind., Ellington Field, Texas.
MILLER, Orval Jerome, Captain, Fort Wayne, Ind., Camp Croft, S. C.
MINNIG, Donald Irwin, 1st Lieut., Akron, Ohio, Camp Shelby, Miss.
MONTGOMERY, Edward Lee, 1st Lieut., Circleville, Ohio, Camp Shelby, Miss.
MORRIS, Marion Hamilton, 1st Lieut., Indianapolis, Fort Oglethorpe, Ga.
MUELLER, Paul Frederick, 1st Lieut., Lawrenceburg, Ind., Drew Field, Fla.
MUSTA, Walter, 1st Lieut., Macedonia, Ohio, Camp Shelby, Miss.
MYERS, Ben Vernon, 1st Lieut., Elyria, Ohio, Fort Bragg, N. C.
NAUMAN, John Henry, Captain, Martin's Ferry, Ohio, Camp Forrest, Tenn.
NEWPORT, Norsuda Monteville, 1st Lieut., Zanesville, Ohio, Camp Grant, Ill.
OAKES, Milton Caleb, 1st Lieut., Lexington, Ohio, Camp Shelby, Miss.
OSLER, George Edward, 1st Lieut., Newport, Ky., Camp Shelby, Miss.
PATTERSON, Owen Findlay, 1st Lieut., Holmesville, Ohio, Lowry Field, Colo.
PEABODY, Carroll Alden, 1st Lieut., Cleveland, Brookley Field, Ala.
PEEBLES, Thomas Arthur, Major, Lorain, Ohio, Fort Hancock, N. J.
POWELEIT, Alvin Charles, 1st Lieut., Newport, Ky., Fort Knox, Ky.
PRICE, Walter Samuel, 1st Lieut., Phillipsburg, Ohio, Camp Forrest, Tenn.
RALSTON, James Gilbert, 1st Lieut., Clarksburg, W. Va., Camp Claiborne, La.
RAMSEY, Hugh Smith, 1st Lieut., Bloomington, Ind., Fort Oglethorpe, Ga.
RAMSAYER, Herbert Perry, 1st Lieut., Canton, Ohio, Camp Shelby, Miss.
RANDELL, Asher, 1st Lieut., Youngstown, Ohio, Fort Bragg, N. C.
RARICK, Alden Jasper, Captain, Cromwell, Ind., Camp Lee, Va.
RATH, Albert Ernest, 1st Lieut., Wooster, Ohio, Fort Sam Houston, Texas.
REHBOCK, Donald Jacob, Captain, Cleveland, Army Medical Museum, Washington, D. C.
RIEBEL, John Alan, II, 1st Lieut., Ashland, Ohio, Langley Field, Va.
ROBERTS, Donald Russell, 1st Lieut., Elkins, W. Va., Fort Bragg, N. C.
ROTHBERG, Maurice, Captain, Fort Wayne, Ind., Fort Oglethorpe, Ga.
RUBELDS, Milton Raymond, 1st Lieut., Indianapolis, Fort Jackson, S. C.
RUNNELS, Scott Clark, Colonel, Cleveland, Camp Shelby, Miss.
SADUGOR, Marvin Gerald, 1st Lieut., Cleveland, Fort Benjamin Harrison, Ind.
SCHERB, Burton Eldon, 1st Lieut., Clay City, Ind., Ellington Field, Texas.
SCHMITT, Richard Klatte, 1st Lieut., Columbus, Ind., Camp Shelby, Miss.
SEDAM, Herbert Lawrence, 1st Lieut., Indianapolis, Fort Knox, Ky.
SEESHOLTZ, John Rinkes, 1st Lieut., Toledo, Ohio, Fort Bragg, N. C.
SENNETT, William Kelsey, 1st Lieut., Monterey, Ind., Fort McClellan, Ala.
SEWELL, Frank Kash, Captain, Jackson, Ky., Fort Screven, Ga.
SHAMANSKY, Harry Solomon, Major, Columbus, Ohio, Fort Buchanan, Puerto Rico.
SHIELDS, Thomas S., 1st Lieut., Indianapolis, Fort Oglethorpe, Ga.
SHORTRIDGE, Wilson Poole, 1st Lieut., Morgantown, W. Va., Pine Camp, N. Y.
SHORTZ, Gerald, 1st Lieut., Kendallville, Ind., Camp Wheeler, Ga.
SIEBENTHAL, Ben Jack, 1st Lieut., Bloomington, Ind., Luke Field, Ariz.
SIGMUND, William Belmer, Captain, Columbus, Ind., Fort Oglethorpe, Ga.
SILVER, Ezra Isreal, 1st Lieut., Cleveland, Fort Jackson, S. C.
SILVER, Francis Friedman, 1st Lieut., Akron, Ohio, Camp Blanding, Fla.
SLICK, Crystal Ray, 1st Lieut., Lynn, Ind., Fort Jackson, S. C.

SMITH, Merl Bernard, 1st Lieut., Toledo, Ohio, Ellington Field, Texas.
SPARKS, Aubrey Leighton, 1st Lieut., Warren, Ohio, Edgewood Arsenal, Md.
SPEED, George William, 1st Lieut., Springfield, Ohio, Fort Buchanan, Puerto Rico.
STEPHENS, Lowell Ralston, Captain, Covington, Ind., Fort Oglethorpe, Ga.
STRATHMAN, William Henry, Captain, Toledo, Ohio, Fort McClellan, Ala.
STUBBINS, William Matthews, 1st Lieut., Elkhart, Ind., Fort Oglethorpe, Ga.
SUTTLE, Robert Courtney, 1st Lieut., Toledo, Ohio, Camp Wheeler, Ga.
TAYLOR, Clifford Chalmer, Captain, Indianapolis, Fort Sam Houston, Texas.
TAYLOR, David, Captain, Yellow Springs, Ohio, Fort Jackson, S. C.
UDELF, Maxwell Spenser, 1st Lieut., Camp Shelby, Miss., Camp Shelby, Miss.
UHRICH, John Henry, 1st Lieut., Fort Wayne, Ind., Air Base, Charlotte, N. C.
URBANSKI, Walter Joseph, 1st Lieut., Cleveland, Camp Blanding, Fla.
VENABLE, Harry W., 1st Lieut., Louisville, Ky., Fort Benning, Ga.
WALES, Homer Louis, 1st Lieut., Indianapolis, Camp Livingston, La.
WALKER, Glenn Harvey, 1st Lieut., Woodville, Ohio, Camp Lee, Va.
WARREN, James Oliver, 1st Lieut., Welch, W. Va., Fort Benning, Ga.
WEAVER, Edgar Stephens, 1st Lieut., Scottsville, Ky., Fort McClellan, Ala.
WEINBLATT, Morris, 1st Lieut., Toledo, Ohio, Fort Hayes, Ohio.
WEISER, Richard Wagner, Captain, Cincinnati, Fort Jackson, S. C.
WHITE, Harvey Eugene, 1st Lieut., Indianapolis, Fort Jackson, S. C.
WILSON, Gerald Newton, 1st Lieut., Columbus, Ohio, Fort Jackson, S. C.
WIRTZ, Robert Edward, 1st Lieut., Canton, Ohio, Kelly Field, Texas.
WOLFRAM, Donald J., Captain, Brownsburg, Ind., Camp Lee, Va.
WOOD, Cyrus Rogers, 1st Lieut., Fort Clinton, Ohio, Fort Jackson, S. C.
WORK, Charles Edwin, 1st Lieut., Fort Thomas, Ky., Borinquen Field, Puerto Rico.
WYTTENBACH, John Edward, Major, Indianapolis, Camp Wheeler, Ga.
ZEIGER, Michael Raymond, Captain, Cleveland Heights, Ohio, Fort Jackson, S. C.
ZENO, Ross Russell, 1st Lieut., Akron, Ohio, Fort Jackson, S. C.
ZWICK, Harold Frederick, 1st Lieut., Decatur, Ind., Fort McClellan, Ala.

SIXTH CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding Officer, Sixth Corps Area, which comprises the states of Wisconsin, Illinois and Michigan:

BELL, Julius Nathaniel, 1st Lieut., Chicago, Reception Center, Fort Sheridan, Ill.
BERGER, Irving Ray, 1st Lieut., Chicago, U. S. Army Examination Station, Chicago.
CUTTS, Robert Irving, 1st Lieut., Chicago, Reception Center, Camp Grant, Ill.
DEUTSCH, Hans, 1st Lieut., Chicago, U. S. Army Induction Station, Chicago.
HAMMER, John M., 1st Lieut., Kalamazoo, Mich., 1605th Corps Area Service Unit, Fort Custer, Mich.
HUNT, Homer Hill, 1st Lieut., Ann Arbor, Mich., Corps Area Laboratory, Fort Sheridan, Ill.
JANUS, Arthur Israel, 1st Lieut., Chicago, U. S. Army Recruiting Station, Chicago.
KOCH, Joseph Melchior, 1st Lieut., Granite City, Ill., Reception Center, Scott Field, Ill.
LEPPERT, Charles L., 1st Lieut., Rockford, Ill., Reception Center, Camp Grant, Ill.
MASSOVER, Alfred J., 1st Lieut., Chicago, Reception Center, Fort Sheridan, Ill.
SCHOLLE, Norbert William, 1st Lieut., Muskegon, Mich., U. S. Army Induction Station, Kalamazoo, Mich.
SKOPEK, Frank S., 1st Lieut., Saginaw, Mich., U. S. Army Induction Station, Kalamazoo, Mich.
WEBER, Joseph E., 1st Lieut., Mendota, Wis., U. S. Army Induction Station, Milwaukee.
WEISDORF, William, Captain, Chicago, U. S. Army Induction Station, Chicago.
YANOWITZ, Meyer, 1st Lieut., Chicago, Reception Center, Fort Custer, Mich.

SEVENTH CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, Seventh Corps Area, which comprises the states of North Dakota, South Dakota, Minnesota, Nebraska, Iowa, Kansas, Missouri, Arkansas and Wyoming:

AGNEW, Lloyd Campbell, 1st Lieut., Iowa City, Corps Area Service Command Station Hospital, Fort Des Moines, Iowa.
ALLEN, Marion Bond, 1st Lieut., Cove, Ark., Corps Area Service Command Induction Station, Jefferson Barracks, Mo.
BASKIN, Abraham Hyman, 1st Lieut., Lincoln, Neb., Corps Area Service Command Station Hospital, Fort Riley, Kan.
BERNERT, Henry Schelten, 1st Lieut., Kansas City, Mo., Corps Area Service Command Station Hospital, Fort Leonard Wood, Mo.
FLEMING, Jacob William, Jr., 1st Lieut., Moberly, Mo., Corps Area Service Command Station Hospital, Fort Leonard Wood, Mo.
HOGAN, Clifford William, 1st Lieut., Kensington, Minn., Corps Area Service Command Station Hospital, Fort Snelling, Minn.
LUEDDE, Philip Shryock, Captain, St. Louis, Corps Area Service Command Station Hospital, Camp J. T. Robinson, Ark.
McCUISTION, Columbus H., Jr., 1st Lieut., Rochester, Minn., Corps Area Service Command Station Hospital, Fort Snelling, Minn.
MEYER, Alfred Charles, 1st Lieut., Rochester, Minn., Corps Area Service Command Station Hospital, Fort Snelling, Minn.

Orders Revoked

DALTON, Marvin Lewis, 1st Lieut., Brinkley, Ark., Corps Area Service Command Station Hospital, Camp J. T. Robinson, Ark.
HALPERIN, Philip H., 1st Lieut., Kansas City, Mo., Corps Area Service Command Station Hospital,, Fort Leavenworth, Kan.
JOHNSON, Clive Roland, 1st Lieut., Rochester, Minn., Air Corps Station, McChord Field, Tacoma, Wash.
JONES, Otey S., Captain, St. Louis.
McCOMAS, Marmaduke D., Captain, Courtland, Kan.
NEUMAIER, Arthur, 1st Lieut., Glencoe, Minn.
SMITH, Louis J., 1st Lieut., New Madrid, Mo.
SMRHA, James A., 1st Lieut., Cedar Rapids, Iowa.
SPIELHAGEN, Guenther F., 1st Lieut., Iowa City.
TAMISIEA, Francis X., 1st Lieut., Missouri Valley, Iowa.
WINDER, Clifford D., Captain, Waterloo, Iowa.

Relieved from Active Duty

ABRAMSON, Milton, Captain, Minneapolis, Fort Snelling, Minn.
AGNEW, James Ward, Captain, Iowa City, Fort Des Moines, Iowa.
FREEDMAN, Albert L., 1st Lieut., Iowa City, Fort Des Moines, Iowa.

EIGHTH CORPS AREA

The following additional medical reserve corps officers have been ordered to active duty by the Commanding General, Eighth Corps Area, which comprises the states of Colorado, Arizona, New Mexico, Oklahoma and Texas:

DIENGER, Bernard C., 1st Lieut., Winslow, Ariz., 55th Medical Battalion, Fort Sam Houston, Texas.
SHAPIRA, Jake, 1st Lieut., Conroe, Texas, 45th Division, Camp Barkeley, Texas.
TRITT, Earl F., Captain, Dulce, N. M., Station Hospital, Camp Wallace, Texas.

Relieved from Active Duty

GORDON, Everett J., Captain, 46th Bombardment Squad, 41st Bombardment Group, Tucson, Ariz.
NICHOLS, Ace Elliott, Captain, Gulf Coast Air Corps Training Center, Randolph Field, Texas.
PETERS, Isadore D., 1st Lieut., Station Hospital, Camp Wallace, Texas.
RAY, John Wyeth, 1st Lieut., 64th Medical Regiment, Camp Bowie, Texas.

ORDERED TO FOREIGN DUTY

ASHTON, Paul Louis, 1st Lieut., Headquarters, 12th Medical Regiment.
BERNARDINI, Camillo Victor, Major, Station Hospital, Sitka, Alaska.
KRANSON, Seymour Julian, Captain, Clayton, Mo., Tripler General Hospital, Honolulu, Hawaii.
MORRISON, Marcus Eugene, 1st Lieut., Columbus, Ohio, Tripler General Hospital, Honolulu, Hawaii.
OLSSON, Paul Bryant, 1st Lieut., Station Hospital, Fort Greeley, Kodiak, Alaska.
RING, Harold Henry, Captain, Tripler General Hospital, Honolulu, Hawaii.
SAUNDERS, George Chancellor, 1st Lieut., Station Hospital, Fort Richardson, Alaska.
SCHENTHAL, Joseph Edwin, 1st Lieut., Station Hospital, Fort Amador, Canal Zone.
SCHNEIDER, Louis W., Captain, Montgomery, Ala., Clark Field, Fort Stotsenburg, Philippine Islands.
SHAW, Vaughan Allison, 1st Lieut., Houlton, Maine, Fort Stotsenburg, Philippine Islands.
STARK, James Andrew, 1st Lieut., Station Hospital, Fort Richardson, Alaska.
TOPP, Olfert Winther, 1st Lieut., Greenville, S. C., Borinquen Field, Puerto Rico.
VAN DEVENTER, William Clarke, Captain, N. G., Medical Detachment, 250th Coast Artillery, Dutch Harbor, Alaska.

ORGANIZATION SECTION

OFFICIAL NOTES

ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE

The Thirty-Eighth Annual Congress of the Council on Medical Education and Hospitals of the American Medical Association, cooperating with the Federation of State Medical Boards of the United States, will be held at the Palmer House, Chicago, Feb. 16 and 17, 1942. A preliminary announcement of the program follows:

The Effect of the National Defense Program on Medical Practice Among the Civilian Population and on the Maintenance of Accepted Standards in Medical Education and the Cultivation of Research.
Ray Lyman Wilbur, M.D., Chairman, Council on Medical Education and Hospitals, Stanford University, Calif.

The Relation of the Chemist to Medicine.
Vincent du Vigneaud, Ph.D., Professor of Biochemistry, Cornell University Medical College, New York.

(Topic to be Supplied.)
Rufus C. Harris, LL.D., President, Tulane University of Louisiana, New Orleans.

The Relation of Pharmacology to Therapy.
Soma Weiss, M.D., Hersey Professor of the Theory and Practice of Physic, Harvard Medical School, Boston.

Federal Aid to Education.
William P. Munro, Ph.D., LL.D., Professor of History and Government, California Institute of Technology, Pasadena.

The Effect of the War on Medical Education in Canada.
J. C. Meakins, M.D., Dean, McGill University Faculty of Medicine, Montreal, Quebec.

Current Medical Personnel Problems of the Army.
Col. George F. Lull, M.D., Chief, Personnel Division, United States Army Medical Corps, Washington, D. C. (Representing the Surgeon General of the United States Army).

Medical Education from the Standpoint of the Navy Medical Corps.
Rear Admiral Ross T. McIntire, Surgeon General, United States Navy, Washington, D. C.

Needs of the Public Health Service for Medical Personnel in National Defense Activities.
Thomas Parran, M.D., Surgeon General, United States Public Health Service, Washington, D. C.

Selective Service and Medical Students.
Brig. Gen. Lewis B. Hershey, Director, Selective Service System, Washington, D. C.

The Committee on Medical Preparedness of the American Medical Association.
Irvin Abell, M.D., Chairman, Committee on Medical Preparedness of the American Medical Association and Chairman, Health and Medical Committee of the Federal Security Agency, Louisville, Ky.

Medical Education in the Preparedness Program.
Harold S. Diehl, M.D., Dean, University of Minnesota Medical School, Minneapolis, and Member, Procurement and Assignment Service for Physicians, Dentists and Veterinarians of the Office of Defense, Health and Welfare Services.

Access to Hospital Records—Some Legal Aspects.
T. V. McDavitt, Bureau of Legal Medicine and Legislation, American Medical Association, Chicago.

The Function of the State Hospital as an Educational and Social Agency.
Winfred Overholser, M.D., Medical Superintendent, St. Elizabeths Hospital, Washington, D. C.

Summary of the Work of the Council on Industrial Health of the American Medical Association.
S. J. Seeger, M.D., Chairman, Council on Industrial Health, American Medical Association, Texarkana, Texas.

The Integrated Course in Anatomy as Related to Licensure.
B. I. Burns, M.D., Dean, Louisiana State University School of Medicine, New Orleans.

Teaching of Professional Ethics in Medical Schools.
Arthur T. McCormack, M.D., State Health Commissioner, Louisville, Ky.

Citizenship as Related to Licensure.
Walter E. Vest, M.D., President, Public Health Council, West Virginia Department of Health, Huntington.

Accelerating Medical Training in Canada.
E. Stanley Ryerson, M.D., Assistant Dean, University of Toronto Faculty of Medicine, Toronto, Ontario.

Industrial Health as Related to Licensure.
C. O. Sappington, M.D., Executive Director, American Conference on Industrial Health, Chicago.

Licensure Problems in the District of Columbia.
George C. Ruhland, M.D., Secretary-Treasurer, Commission on Licensure, Washington, D. C.

Have Basic Science Laws Advanced the Practice of Medicine?
Thomas J. Crowe, M.D., Secretary, Texas Board of Medical Examiners, Dallas.

Revision of the Constitution and By-Laws of the Federation of State Medical Boards.
T. V. McDavitt, Bureau of Legal Medicine and Legislation, American Medical Association, Chicago.

Morris Fishbein, M.D., Editor, THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, Chicago, will be the guest speaker at the Annual Dinner of the Federation of State Medical Boards on Monday, February 16. His topic will be:

American Medicine and the National Emergency.

THE ATLANTIC CITY SESSION

Motion Pictures in the Scientific Exhibit

The Committee on Scientific Exhibit is arranging a motion picture program for the Atlantic City session. Motion pictures will be shown only in theaters provided for the purpose rather than in the exhibitor's booth. Six or more such theaters will show pictures simultaneously and continuously throughout the week.

Application blanks for a place on the motion picture program may be obtained from the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago.

MEDICAL LEGISLATION

MEDICAL BILLS IN CONGRESS

Changes in Status.—S. 165 has passed the Senate and House, providing for continuing in the service of the Army, Navy, Marine Corps and Coast Guard beyond the term of their enlistment those suffering from service connected diseases or injury who are in need of further medical care or hospitalization. S. 1826 has passed the Senate and House, permitting seeing-eye dogs to enter government buildings when accompanied by their blind masters.

Bills Introduced.—H. Res. 337, introduced by Representative Celler, New York, proposes to create a committee of five members of the House of Representatives to study, investigate and report to the House on the proposals that have been made for amendment of the Social Security Act, including a proposal to increase the number of persons to be embraced within the benefits of that act so as to include domestics, farm operators, agricultural workers, employees in nonprofit institutions, casual laborers, employees of state and local governments, professional men and women, and other self-employed persons. H. J. Res. 250, introduced by Representative Bloom, New York, proposes to grant permission to Thomas Parran, Surgeon General of the United States Public Health Service, Bolivar J. Lloyd, Medical Director (retired), Howard F. Smith, Medical Director, Herbert A. Spencer, Medical Director, and Gilbert L. Dunnahoo, Surgeon, all of the United States Public Health Service, to accept and wear certain decorations bestowed on them by the governments of France, Cuba, Chile, Finland and Luang-Prabang. H. R. 6029, introduced by Representative Knutson, Minnesota, H. R. 6055, introduced by Representative Reece, Tennessee, H. R. 6111, introduced by Representative Tibbott, Pennsylvania, and H. R. 6136, introduced by Representative Maciejewski, Illinois, propose to amend section 2800 of the Internal Revenue Code, to authorize the Commissioner of

Internal Revenue to make a refund of the distilled spirits tax, at the rate of $1 per gallon, to any person who has used distilled spirits, produced in a domestic registered distillery or industrial alcohol plant, in the manufacture or production of an article intended for use for nonbeverage purposes, subject to certain limitations. H. R. 5827, introduced by Representative Sauthoff, Wisconsin, proposes to require a mental examination of the accused prior to trial of any charge by Army general court martial. H. R. 6030, introduced by Representative Kunkel, Pennsylvania, proposes to amend an act to provide books for the adult blind by increasing the annual appropriation from $275,000 to $350,000, by authorizing the purchase of periodicals in addition to books, and by providing that not less than $2,500 of the sum allocated for books and periodicals in raised characters shall be expended for the publication in braille, for the exclusive use of the deaf-blind, of the magazine of the American League for the Deaf-Blind known as the *Bulletin Board*. H. R. 6069, introduced by Representative Hook, Michigan, provides that after March 11, 1941 all members of the armed forces of the United States shall be entitled to the same benefits awarded to members of the armed forces of the United States as under the World War Veterans' Act of June 7, 1924. H. R. 6106, introduced (by request) by Representative Rankin, Mississippi, proposes to provide liberalized benefits for disabled American veterans of the World War and their dependents. This bill, among other things, provides that in the administration of laws pertaining to veterans, retired officers and enlisted men of the Army, Navy, Marine Corps and Coast Guard, who served honorably during a war period as recognized by the Veterans' Administration, shall be entitled to hospitalization and domiciliary care in the same manner and to the same extent as veterans of any war are now or may hereafter be furnished hospitalization or domiciliary care by the Veterans' Administration. H. R. 6135, introduced by Representative Lanham, Texas, proposes to authorize an additional appropriation of $150,000,000 to construct community facilities in defense areas, including hospitals, health centers and clinics. H. R. 6138, introduced by Representative Clason, Massachusetts, provides that any person claiming to be a citizen of the United States at birth, in whose case no official record of birth is available, may apply to the Commissioner of Immigration and Naturalization for a certificate of citizenship.

DISTRICT OF COLUMBIA

Change in Status.—H. R. 5694 has passed the House and has been reported to the Senate, proposing to prevent the sale of unwholesome food in the District of Columbia. A similar bill, S. 2045, was introduced by Senator McCarran, Nevada.

Bill Introduced.—H. R. 6097, introduced by Representative D'Alesandro, Maryland, provides that the National Hospital Service Society, Incorporated, shall be considered to be a fraternal organization and shall be permitted to do business without further license or permit, exempt from the provisions of the insurance laws relating to the District of Columbia.

MEDICAL ECONOMIC ABSTRACTS

MEDICAL CHANGES IN BRITAIN

Bombing and overcrowded insanitary shelters led the British to anticipate epidemics, says Ritchie Calder in *John O'London's Weekly* (April 25, 1941). But when a body of American experts under Prof. J. E. Gordon of Harvard arrived they found that the expected epidemic had failed to materialize.

> In fact, the shelters had a lower disease rate than the average for the country. And the country as a whole, apart from the war epidemic of "spotted fever," or cerebrospinal meningitis, due mainly to the bringing together of young people under dormitory conditions, in the army and other ways, could show a bill of health comparable with the good years immediately before the war.

Although the war has not yet brought epidemics, it has created problems in the organization of medical services. There have been many proposals for changes in the panel system, which this writer says "no one would dare to pretend . . . has been satisfactory." It is generally agreed that the "approved society" system is wasteful. The British Medical Association has proposed to extend the panel system to dependents, but the first effect of this would be to make nearly double the sales value of existing practices, which would compel physicians to pay some $10,000 to start in practice.

The voluntary hospital system has been heading for bankruptcy for several years—a fate which increased gifts and hospitalization insurance have only postponed.

> As a result of the war, some of these hospitals began, for the first time in history, to bank money. They were paid by the state to keep the beds empty for casualties. The government leased these beds at anything from two and a half guineas to seven guineas a week (about $8 to $25). The hospitals were then able to lease emergency hospitals, built by the state, with the help of the bed rent from the state. This paradoxical device maintained the façade of the voluntary system. And since the war began, hospitals have been among the main targets of the Nazi air raiders, and an essential part of the postwar replanning will have to deal with their rebuilding.

Private practice has been hard hit. Large numbers of doctors have been recruited for the forces and others taken into the government's hospital scheme. Expansion of industrial practice and special shelter service have taken others, and evacuations and the creation of "defense areas" have caused widespread dislocations. It has also brought some steps toward reorganization and numerous proposals for reconstruction after the war. A "regional plan" for hospitals to include both voluntary and government operated hospitals has been developed, and there seems to be a trend toward state medicine with salaried physicians as part of an integrated system.

Manifestly the old order in medicine is finished.

> One thing is certain: We shall have the right to demand after this not the sickness service, which is all we have got at the moment, but a health service, embracing the whole population, irrespective of income, and providing medical care from birth to death, not as a purchasable commodity, but as an inalienable right. That service would conceive health as a biological trinity—protective medicine, which includes nutrition and environment (positive health); preventive medicine, which is the public health services, our pickets against disease; and curative medicine, including early diagnosis and highly skilled treatment, with all the resources of modern medical science and practice.

COMPULSORY SICKNESS INSURANCE IN CALIFORNIA

The proposal to secure signatures for a referendum for compulsory sickness insurance in California received a setback when the members of the influential Commonwealth Club voted opposition to any such system of sickness insurance.

To the question "Do you favor the establishment by government of a system of compulsory sickness insurance (health insurance)?" eight hundred and seventy-one members voted No and four hundred and ninety-one members voted Yes.

Ballots on this question were mailed to all of the Club's four thousand four hundred members following a study by the Public Health Section and publication of pro and con reports on the issue.

TUBERCULOSIS MORTALITY

In 1900 there were 200 deaths per hundred thousand annually from tuberculosis in the United States, according to the statisticians of the Metropolitan Life Insurance Company. In 1921 that rate had been halved and in 1940 the reports now available indicate that it will be more than halved again, since it will be a little less than 45 per hundred thousand. However, the rate of decrease seems to be slowing up. The year 1936 even showed a slight increase. Contrary to expectations, the rate of decline was a little faster during the greatest depression years than in some of the subsequent years. In 1940, twelve states had death rates from tuberculosis below 30 per hundred thousand and six of 20 or less per hundred thousand.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

CALIFORNIA

Supervisor of Student Health Service.—Dr. William J. Norris has been appointed university physician and will take general charge of the student health service on the Los Angeles campus of the University of California. He will not replace Dr. Donald S. MacKinnon, physician for men, or Dr. Lillian R. Titcomb, physician for women, according to the University *Clip Sheet*. Dr. Norris was formerly connected with the Los Angeles campus, serving as physician for men from 1921 to 1932. He graduated at Columbia University College of Physicians and Surgeons, New York, in 1918. The *Clip Sheet* stated that the appointment of Dr. Norris anticipates the building of a $1,000,000 Student Health Center and Hospital on the Los Angeles campus.

Dr. Housman Resentenced to San Quentin. — Superior Judge Lile T. Jacks sentenced Dr. Nathan S. Housman, San Francisco, on October 24, to serve from one to fourteen years in San Quentin, on conviction of perjury and offering and preparing false evidence in connection with an earlier trial for failure to keep proper records of narcotic prescriptions. This judgment was a resentence, confirming an earlier sentence on three counts to run concurrently. Dr. Housman was first sentenced on June 13, 1940 after the court had denied motion for a new trial. He had been indicted Nov. 1, 1939 on six counts: two of perjury, two of preparing false evidence and two of offering false evidence. He appealed to the state appellate court and then the state supreme court. His appeal to the U. S. Supreme Court to review his case failed.

COLORADO

Place of Annual Session. — The Colorado State Medical Society has announced that its seventy-second annual session will be held in Colorado Springs, Sept. 23-26, 1942.

Industrial Hygiene Division. — The Colorado industrial hygiene unit, formerly a section of the division of sanitary engineering, has been established as a separate division of the state board of health, it was announced in September. Mr. R. J. Owens is director of the new division.

The Friedman Lectures.—The William S. Friedman Lectures, sponsored by the National Jewish Hospital, Denver, in cooperation with the Denver County Medical Society and the University of Colorado School of Medicine, Denver, were delivered in November by Drs. Jesse G. M. Bullowa, clinical professor of medicine, New York University College of Medicine, New York,, and Leo Eloesser, clinical professor of surgery, Stanford University School of Medicine, San Francisco. Dr. Bullowa spoke on "Chemotherapy in the Pneumonias and the Immunity Reactions" and Dr. Eloesser on "Causes of Cavitation in Pulmonary Tuberculosis." At a special meeting of the Medical Society of the City and County of Denver on November 18 Dr. Eloesser discussed "Compound Fractures in War Time" and Dr. Bullowa "Pneumonias in the Infectious Diseases of Childhood." Both men conducted clinics. Local physicians also participated in the program.

ILLINOIS

State-Subsidized Tumor Diagnostic Service.—The second state-subsidized tumor diagnostic service has been established at the Methodist Hospital of Central Illinois, Peoria, with the approval of the Peoria County Medical Society. The first was begun at the Memorial Hospital of Springfield on October 1. Plans for a tissue diagnostic service at the Marshall Browning Hospital, Du Quoin, have been approved by the Perry County Medical Society and it is expected that this unit will soon be in operation. Although the program for this diagnostic service is administered by Dr. Raymond V. Brokaw, chief of the division of cancer control of the state department of health with headquarters in Springfield, the actual management of the services will be the responsibility of the selected hospitals which are chosen by the local medical society. These tumor diagnostic services are designed to provide for the general practitioner a competent consultation service without cost for his suspected cancer cases. The facilities will be available to every physician for the reference of his case for diagnosis and recommendation as to treatment, the patient being returned to the physician for further action. No treatment is to be rendered by this service in any case. The tissue diagnostic service provided for by the 1939 legislature was inaugurated in September 1940 as a function of the division of laboratories of the health department at 1800 West Fillmore Street, Chicago. The act creating the division of cancer control provided for an advisory board of seven members to be appointed by the governor. To make these appointments "the governor shall invite nominations from recognized medical organizations of this state," and no less than four members should be recognized authorities in the cancer field, and at least four should be physicians. At present the board consists of Drs. David J. Davis, Chicago, chairman; William M. Cooley, Peoria; Fauntleroy Flinn, Decatur; Roswell T. Pettit, Ottawa; James S. Templeton, Pinckneyville; John A. Wolfer, Chicago, and Edwin F. Hirsch, Chicago, secretary.

Chicago

Symposium on Old Age.—The Chicago Medical Society devoted its December 10 meeting to a symposium on old age. Speakers were Edmund V. Cowdry, Ph.D., St. Louis, on "The Problem of Aging"; Dr. Wilber E. Post, "Medical Management of the Aging Patient"; Dr. Lester R. Dragstedt, "The Aged Patient as a Surgical Risk," and Dr. Francis J. Gerty, "Psychologic Problems in the Aging Patient."

Annual Surgical Prize. — The Chicago Surgical Society announces that competition for the 1942 annual prize is now open to young men devoting themselves to surgery in Cook County and who are not members of the society. A prize of $250 will be awarded for the most meritorious original investigation in one or both of the fields of experimental and clinical surgery. The paper submitted should be of original work which has not been printed or presented previously. The manuscript should bear no identification marks of author, hospital or institution but should be accompanied by a sealed envelop bearing on its outside the title of the paper and containing within the name and address of the author. No award will be made if no paper submitted is deemed worthy. Manuscripts should be sent to the secretary of the Chicago Surgical Society, 54 East Erie Street, not later than March 1.

INDIANA

Personal.—Dr. Charles P. Anderson, assistant superintendent of the health department in Durham, N. C., has been appointed director of the School Medical Service of Gary.—— Dr. Robert A. Nason, Garrett, has been appointed coroner of DeKalb County, filling the unexpired term of the late John R. Clark, Auburn.——Dr. Harley F. Flannigan, Lagrange, has been named health commissioner of Lagrange County, succeeding Dr. Frank Morse Nichols, Topeka, resigned. Dr. Nichols and his family are moving to Warsaw.

District Meeting. — The Third District Medical Society was addressed in French Lick, November 5. Dr. Carl P. Huber, Indianapolis, opened the session with a discussion of "Normal Delivery, Episiotomy, Forceps and Cesatean Section." Other speakers were Drs. Minor W. Miller, Evansville, on syphilis; Arthur F. Weyerbacher, Indianapolis, gonorrhea; R. Hayes Davis, Louisville, pneumonia; Russell A. Sage, Indianapolis, ear infections; Wendell D. Little, Indianapolis; Louis H. Segar, Indianapolis, rheumatic fever; George J. Garceau, Indianapolis, fractures; Louis Wallace Frank, Louisville, peptic ulcer, and James O. Ritchey, Indianapolis, the after dinner speaker, "Endocrine Disorders."

KANSAS

Rehabilitation Program Begun.—The Kansas State Board of Health, Topeka, is sponsoring a program of rehabilitation of selective service registrants who have been rejected for military service by reason of physical disabilities. Statistical information on registrants who are afflicted with diseases of the lungs and venereal disease is now being assembled, the program to be extended into other fields when facilities permit.

Society News. — The Wyandotte County Medical Society will be addressed, December 16, at its annual meeting by Dr. James W. May on "Common Tumors of the Eye." Dr. Tom R. Hamilton spoke on "Blood Cultures in Clinical Pathology" at the society's meeting December 2 and Dr. Ward W. Summerville, "The Friedman Test." All are from Kansas City.

MINNESOTA

Society News.—Karl F. Meyer, Ph.D., of the George Williams Hooper Foundation for Medical Research, University of California, San Francisco, will address the Minnesota Pathological Society in Minneapolis, December 16, on "The Animal Kingdom, a Reservoir of Infection." The society was addressed, November 18, by Melvin H. Knisely, Ph.D., Chicago, on "Effects of Plasmodium Malariae on the Blood Vascular System."

Medals Awarded.—Dr. Edward H. Juers, Red Wing, recently received the annual medal of the Southern Minnesota Medical Association for his paper on epiploitis. The medal is given to the author of the paper considered best on the program of the association's annual meeting. The medal for a case history presented on the annual program went to Dr. Peter E. Hermanson, Hendricks, whose report dealt with a case of pregnancy occurring outside the uterus.

NEVADA

District Meeting of Indian Service.—The U. S. Indian Service conducted a district medical meeting for California, Nevada and Utah in Reno, November 12-13. Included among the speakers were:

Dr. Ralph B. Snavely, San Francisco, Indian Health and National Defense.
Dr. Fred T. Foard, San Francisco, Defense Created Public Health Needs in the Western States.
Dr. Hubert V. Hailman, Salt Lake City, Trachoma, the Enigma.
Dr. Horace De Lien, San Francisco, Case Finding in Tuberculosis.
Dr. Michel Pijoan, Owyhee, A Study of Vitamin Deficiencies at Western Shoshone.
Dr. Roland W. Stahr, Reno, Role of Histamine in Allergy.
Mr. Don C. Foster, superintendent, Carson Indian Agency, Stewart, History of Development of Indian Service in Nevada.
Mr. W. W. White, sanitary engineer, Nevada State Health Department, Reno, Sanitary Engineering Aspects of Communicable Disease Control.

NEW JERSEY

Society News.—Dr. Irving S. Wright, New York, will discuss "Peripheral Vascular Diseases" before the Bergen County Medical Society, Hackensack, December 9.——The Passaic County Medical Society devoted its meeting in Paterson on November 13 to a discussion of "State Medicine—Its Trends." The speakers were Drs. Thomas K. Lewis, Camden, and Leroy A. Wilkes, Trenton, president and executive officer respectively of the Medical Society of New Jersey.—— Dr. Aaron H. Neffson, New York, addressed the Monmouth County Medical Society in Matawan, November 26, on "Management of Acute Laryngotracheobronchitis.—— The Camden County Medical Society was addressed in Camden, December 2, on "Surgical Therapy in Lesions of the Large Bowel."

NEW YORK

Rochester Academy Rededicates Museum.—The Rochester Academy of Medicine, Rochester, rededicated its medical museum to Dr. John R. Williams, chairman of the museum commission, November 16. A reception to guests opened the program and speakers included Dr. William J. Merle Scott, president of the academy; Dr. George H. Whipple, professor of pathology and dean, University of Rochester School of Medicine and Dentistry; Dr. Walter B. Cannon, George Higginson professor of physiology and head of the department, Harvard Medical School, Boston, and Dr. Williams. The museum and auditorium are located in a new wing adjoining the academy's home, which was the gift in 1938 of the daughters of E. P. Lyon in memory of their parents. A feature of the medical museum is a memorial frieze in which is recognized the fundamental contributions made by twenty-four North American scientists in the past hundred and fifty years. Those honored in the frieze are:

Dr. Benjamin Rush, Philadelphia.
Dr. William Beaumont, St. Louis.
Dr. Crawford W. Long, Athens, Ga.
Dr. William T. G. Morton, New York.
Dr. Oliver Wendell Holmes, Cambridge, Mass.
Dr. Ephraim McDowell, Danville, Ky.
Dr. Silas Weir Mitchell, Philadelphia.
Dr. Edward L. Trudeau, Saranac Lake, N. Y.
Dr. Theobald Smith, Princeton, N. J.
Dr. Howard T. Ricketts, Chicago.
Dr. Hans Zinsser, Boston.
Dr. Harvey Cushing, New Haven, Conn.
Lafayette B. Mendel, LL.D., New Haven, Conn.
Elmer V. McCollum, Sc.D., Baltimore.
Dr. William C. Gorgas, Washington, D. C.
Dr. Walter Reed, Washington, D. C.
Dr. Abraham Jacobi, New York.
Dr. William Osler, Oxford, England.
Dr. Walter B. Cannon, Boston.
Dr. James B. Herrick, Chicago.
Dr. Frederick G. Banting, Toronto.
Dr. Charles H. Best, Toronto.
Dr. George R. Minot, Boston.
Dr. Whipple.

Another feature is a mural in which is portrayed the contributions of medicine to modern civilization. There are in the museum twenty-three glass cases of priceless memorabilia. Special guests at the dedication included relatives of the honored scientists. In 1936 the medical museum of the Rochester Academy of Medicine was organized as a division of the Rochester Museum of Arts and Sciences.

New York City

Annual Fraternity Lecture.—The Tau chapter of Nu Sigma Nu will offer its annual open lecture, December 15, in the auditorium of Cornell University Medical College. Dr. George J. Heuer, professor of surgery at Cornell will deliver the lecture on "William S. Halsted."

Committee for Russian War Relief.—A Medical Center Committee to Aid Russian War Relief was recently organized with Dr. Tracy J. Putnam, director of the Neurological Institute of New York, as chairman; Dr. Alwin M. Pappenheimer, professor of pathology, Columbia University College of Physicians and Surgeons, treasurer, and Elvin A. Kabat, Ph.D., of the Neurological Institute, secretary. Members of the staffs of the various units forming the Medical Center at 168th Street and Broadway are included in the membership of the committee, which has for its purpose the collection of medical supplies and instruments for the relief of the Russian people.

Ophthalmologists Honored.—Dr. John E. Weeks, professor of ophthalmology emeritus, New York University College of Medicine, was guest of honor at a dinner, October 6, attended by eighty friends and students. Dr. Daniel B. Kirby, professor of ophthalmology at the university, was toastmaster. At the dinner it was announced that the Eye Surgery Fund, a local corporation, had been dedicated to the late Dr. Webb W. Weeks, professor of ophthalmology. The fund is now directed by a medical board consisting of certain members of the faculty of the department of ophthalmology of New York University College of Medicine and of the staff of the department of ophthalmology of Bellevue Hospital. It aims to promote and aid research in surgery of the human eye, disseminate knowledge in this field among physicians and particularly to perform such work as may aid the indigent in need of eye surgery or medical care. While the scope of the fund is now local in character, it is hoped to extend it to a national basis.

NORTH CAROLINA

Dr. Royster Observes Seventieth Birthday.—The Raleigh Academy of Medicine was entertained at a dinner November 19, by Dr. Hubert A. Royster to celebrate his seventieth birthday. Dr. Royster is a descendant of one of the founders of the academy, Dr. Wisconsin I. Royster. The speakers included Drs. Chauncey L. Royster, Raleigh, nephew of the host, on "Early Diagnosis of Shock." Two sons, Dr. Henry P., Philadelphia, and Hubert A. Jr., Bryn Mawr, Pa., spoke on "Nutrition in Surgical Patients" and "Resuscitation of the Newborn," respectively. Dr. Royster was born in Raleigh on Nov. 19, 1871. He graduated at the University of Pennsylvania School of Medicine, Philadelphia, in 1894. He was dean and professor of gynecology at the University of North Carolina Medical Department, Raleigh, from 1902 to 1910 and since 1935 has been professor of surgery at Wake Forest College, now known as the Bowman Gray School of Medicine of Wake Forest College, Winston-Salem. He was president of the Raleigh Academy of Medicine in 1905, of the Tri-State Medical Association in 1906 and the North Carolina State Medical Society in 1921-1922. Dr. Royster served as chairman of the Section on Surgery, General and Abdominal, of the American Medical Association in 1931-1932.

OHIO

Society News.—Dr. Gordon F. McKim, Cincinnati, addressed the Montgomery County Medical Society in Dayton recently on "Infections of Upper Urinary Tract."——Dr. Claude S. Beck, Cleveland, discussed "Heart Disease Treated by Operation" before the Mahoning County Medical Society, November 18.

Henry Freiberg Memorial.—*The Journal of Medicine* for November was dedicated to the memory of Dr. Henry B. Freiberg, who at the time of his death in January was assistant professor of clinical surgery (urology) at the University of Cincinnati College of Medicine. Dr. Freiberg was at one time secretary of the Cincinnati Academy of Medicine which, with other societies, publishes its proceedings in the *Journal of Medicine*.

Commission for the Blind.—Dr. Albert D. Frost, Columbus, has been elected president of the Ohio Commission for the Blind. Other members are Walter Schmitt, Cincinnati; Henry J. Robison, chief of the division of social administra-

tion, state department of public welfare, and Byron P. Redman, Columbus; Mrs. J. D. Johnson, Celina, and W. G. Scarberry, superintendent of the Ohio State School for the Blind, Columbus.

PENNSYLVANIA

Academy of Physical Medicine. — The Pennsylvania Physical Therapy Association has changed its name to the Pennsylvania Academy of Physical Medicine, according to an announcement from Dr. Alfred J. M. Treacy, Philadelphia, secretary. At its meeting, November 21, the group was addressed by Drs. William H. Schmidt, Philadelphia, on "Treatment of Peripheral Vascular Diseases"; Albert A. Martucci, Philadelphia, "Treatment of Anterior Poliomyelitis," and Oscar T. Wood Jr., Philadelphia, "Treatment of Painful Conditions of the Shoulder."

Philadelphia

British Psychiatrist Gives Weir Mitchell Oration. — Dr. Robert D. Gillespie, London, chief psychiatrist of the British Royal Air Force, delivered the ninth Weir Mitchell Oration of the College of Physicians of Philadelphia, November 30. His topic was "Psychoneuroses in Peace and War, and the Future of Human Relationships."

Pittsburgh

Annual Scientific Day.—The annual "scientific day" of the Montefiore Hospital Association of Western Pennsylvania was held in Pittsburgh, November 15. The program included two clinical sessions and an address by Dr. Ira I. Kaplan, New York, entitled "What Radiation Can Offer in Medicine and Surgery."

The Renziehausen Memorial Lecture.—Dr. Henry Rawle Geyelin, assistant clinical professor of medicine, Columbia University College of Physicians and Surgeons, New York, delivered the sixth Renziehausen Memorial Lecture at the meeting of the Allegheny County Medical Society, November 18. His subject was "Clinical Aspects of Diabetes Mellitus."

Society News.—Dr. Meredith F. Campbell, New York, discussed "Urinary Tract Obstructions in Infancy and Children" before the Pittsburgh Urological Association, November 10, in cooperation with the Pittsburgh Pediatric Society. —— The Pittsburgh Academy of Medicine devoted its meeting, November 11, to a panel discussion of sulfonamide therapy with the following speakers: Drs. Robert R. Clark, general practice; James M. Strang, internal medicine; Harold G. Kuehner, surgery; Howard H. Permar, pathology; Adelbert Boyd Miller Jr., otolaryngology; James J. Lee, urology, and Theodore O. Elterich, pediatrics.——Among others, Dr. Julian M. Rogoff addressed the Pittsburgh Surgical Society, November 14, on "Surgical Aspects of Adrenal Cortex."

RHODE ISLAND

Personal.—Dr. Peter F. Harrington, Providence, has been appointed director of a new division of tuberculosis control in the Providence health department.

Sectional Meeting of College of Physicians.—A New England sectional meeting of the American College of Physicians will be held at Providence, January 14. A tentative announcement lists clinical sessions at the Peter's House at Rhode Island Hospital, under the supervision of Dr. Charles F. Gormly, and scientific discussions at the auditorium of the Rhode Island Medical Library. A dinner at the Squantum Club will conclude the day's program.

SOUTH CAROLINA

Conference on Student Health.—The second annual South Carolina Conference on Student Health, sponsored by the South Carolina Tuberculosis Committee and the National Student Health Association, was held in Orangeburg, November 28-29. The program included a panel discussion on "Present Status of Health Programs in Our Colleges" and committee meetings devoted to "Control of Tuberculosis and Venereal Diseases in Colleges" and "What Shall Health Instruction Include?" The speakers included Dr. Paul B. Cornely, associate professor of preventive medicine and public health, Howard University School of Medicine, Washington, D. C., on "Implications of Draft Rejections to College Health Programs." Dr. Cornely was guest consultant and adviser during all professional sessions of the conference.

VIRGINIA

Postgraduate Clinic. — The twenty-eighth postgraduate clinic of the University of Virginia Department of Medicine and the department of clinical and medical education of the Medical Society of Virginia was held in Charlottesville, November 14-15. The program consisted of a symposium on gastroenterology and speakers included:

Dr. Porter P. Vinson, Richmond, Va., Diseases of the Esophagus.
Dr. Julian M. Ruffin, Durham, N. C., Present Status of Gastroscopy.
Dr. Chester M. Jones, Boston, Clinical Significance of "Gastritis."
Dr. Thomas T. Mackie, New York, Deficiency States and the Gastrointestinal Tract.
Dr. Warren T. Vaughan, Richmond, Gastrointestinal Manifestations of Allergy.
Dr. Thomas Grier Miller, Philadelphia, Intestinal Intubation.
Dr. William Osler Abbott, Philadelphia, Drugs and the Gastrointestinal Tract.

WASHINGTON

Changes in Health Officers. — Dr. Russell H. Wilson, Bremerton, has been appointed in charge of the Kitsap County health department, succeeding Dr. Harald M. Graning, who was assigned to the Plague Laboratory of the U. S. Public Health Service at San Francisco.——Dr. Emil E. Palmquist, Colfax, health officer of Whitman County, has been granted six months' leave of absence to study health administration at the University of Michigan, Ann Arbor. During his absence Dr. Philip J. Holabach, Colfax, will be health officer.——Dr. Theodore G. Lathrop, White Salmon, has been appointed acting health officer of Klickitat County.

WYOMING

New Head of Venereal Disease Program.—Dr. Nephi H. Savage, director of the division of epidemiology of the Wyoming State Department of Public Health, Cheyenne, has taken over direction of venereal disease problems in the state, according to *Western Public Health*. Private practitioners will be offered a personal consultation service, and a fourth free venereal disease clinic will be opened soon at Laramie, it was announced.

HAWAII

Federal Grant for Hospital Addition.—Queen's Hospital, Honolulu, has been allowed a grant of $250,000 from the Federal Works Agency, Washington, D. C., to finance the construction of a one hundred bed wing. The hospital will provide the funds for furnishings and equipment. An appropriation has also been given to the hospital to increase the school of nursing by twenty-five students, bringing the student roll up to one hundred.

GENERAL

Impostor Claims Relationship to Dr. Moorman.—Dr. Lewis J. Moorman, Oklahoma City, writes that a young woman has been traveling about the country calling on physicians and representing herself either as a patient or as a relative of his. The woman then presents a fraudulent check to pay her bill, getting the remainder of the check in cash. In many instances the woman has used the name Marianne Hunt. The name of Miriam Brown has also been used. Dr. Moorman, in disclaiming any knowledge or relationship, urges physicians to be on the watch for this woman.

Residency in Anesthesiology.—Applications are now being received for appointment of two residents in anesthesiology at the Cook County Hospital, Chicago, in 1942. The appointments will be for three years, with salary and full maintenance, and the training provided will lead to certification by the American Board of Anesthesiology. The residencies are open to recent graduates of approved internships. Dr. Hugh O. Brown, formerly first assistant in the section on anesthesia, blood transfusion and intravenous therapy of the Mayo Clinic, Rochester, Minn., is director of the department of anesthesia at Cook County Hospital.

Russian Government Asks for Books.—An urgent request from the Soviet government for American medical books to supplement the Central Medical Library was received on November 19 at the offices of Russian War Relief, Inc., 535 Fifth Avenue, New York. The list, comprising almost a hundred titles, includes books on brain, spine and neurologic surgery. According to the announcement the Russian government offered to exchange important recent Soviet literature on medicine for some of the more urgently needed volumes in the listing, such as Albee's "Bone Graft Surgery in Disease, Injury and Deformity," Brock's "Injuries of the Skull, Brain and Spinal Cord: Neuropsychiatric, Surgical and Medicolegal" and Hertzler's "Surgical Pathology of Diseases of the Mouth and Jaws."

Conference on Tuberculosis Establishes New Award.— The Mississippi Valley Conference on Tuberculosis at its recent meeting in Columbus, Ohio, presented the first Hoyt E. Dear-

holt Medal to Robert G. Paterson, Ph.D., Columbus, executive secretary of the Ohio Public Health Association. The medal was established last year as a tribute to the late Dr. Dearholt's devotion, service and leadership in tuberculosis work, particularly in the formation of the Mississippi Valley Conference. Dr. Dearholt was for many years executive secretary of the Wisconsin Anti-Tuberculosis Association.

Annual Forum on Allergy. — The fourth Annual Forum on Allergy will be held at the Hotel Statler, Detroit, January 10-11. On Friday preceding the Forum there will be a conducted trip to the allergy clinic of the University of Michigan, Ann Arbor, where the program will be presented by Dr. John M. Sheldon and his staff. Saturday will be given over to a series of study groups at which the speakers will include:

Dr. Karl D. Figley, Toledo, Ohio, Perennial Allergic Coryza.
Dr. Franz L. Blumenthal, Detroit, Contact Dermatitis.
Dr. Theodore L. Squier, Milwaukee, Food Allergy.
Dr. French K. Hansel, St. Louis, Tonsillectomy, Sinus Surgery, and Local Treatment in Nasal Allergy.
Dr. Howard J. Lee, Oshkosh, Wis., Gastrointestinal Allergy, Cyclic Vomiting, and Beginning Migraine.
Dr. Leon Unger, Chicago, Allergic Conjunctivitis.

Saturday evening Dr. Benjamin S. Kline, Cleveland, will discuss "Observations on the Pathology of Allergy" and Dr. Benjamin C. Houghton, Columbus, will show a motion picture on "The Reaction of Sensitized Cells Growing in Vitro to the Appropriate Antigen." Sunday morning will be devoted to symposiums on drug allergy, hay fever and food allergy. At the annual dinner at noon on Sunday the second award of the Forum's Gold Medal for "outstanding contributions to clinical allergy" will be made by Dr. William W. Duke, Kansas City, Mo. Dr. Milton B. Cohen, Cleveland, will make the presentation. Dr. Duke will deliver the annual lecture on "Beginning of Clinical Allergy in the United States." A special lecture will be given by Dr. Harry L. Alexander, professor of clinical medicine and acting head of the department, Washington University School of Medicine, St. Louis, on "Considerations of the Present Status of Clinical Allergy." The session will be concluded with a program entitled "Information on Allergy, Please," aimed to answer any questions not handled in the general program. Features of the general program will include educational exhibits and "The Picture Theater."

Symposium on Relapsing Fever. — Section N of the American Association for the Advancement of Science will devote its meeting, December 30, in the Medical Arts Building in Dallas to consideration of the "Contributions to the Study of Relapsing Fever in the Americas." Dr. Walter H. Moursund, Dallas, will give the historical introduction, other speakers will include:

Dr. Harry E. Wright, Texas State Department of Health, Austin, Texas, Present Distribution, Texas.
Wilton M. Fisher, M.S., instructor in bacteriology and parasitology, Baylor University College of Medicine, Dallas, Tex., Present Distribution in Oklahoma.
M. Dorothy Beck, bureau of epidemiology, state department of public health, San Francisco, Present Distribution, California.
Dr. Herbert C. Clark, director, Gorgas Memorial Laboratory, Panama, R. P., Present Distribution, Panama.
Herbert G. Johnstone, Ph.D., assistant professor of bacteriology, University of California Medical School, San Francisco, Etiology—Taxonomy.
Gordon E. Davis, Sc.D., bacteriologist, Rocky Mountain Laboratory, U. S. Public Health Service, Hamilton, Mont., Species Unity or Plurality of the Spirochete. Tick Vectors and Life Cycles of Ticks.
Malcolm H. Soule, LL.D., professor of bacteriology, University of Michigan Medical School, Ann Arbor, Mich., Cultivation of Spirochetes.
Robert A. Cooley, Sc.D., Rocky Mountain Laboratory, U. S. Public Health Service, at Hamilton, Determination of Ornithodorus Species.
Dr. Edward Francis, National Institute of Health, U. S. Public Health Service, Washington, D. C., Longevity of Fasting Ornithodorus Tunicata.
Charles M. Wheeler, Ph.D., Hooper Foundation, University of California Medical School, San Francisco. The Distribution of the Spirochetes within the Body of the Vector.
Dr. Harlin L. Wynns, state department of public health, San Francisco, Epidemiology.
Dr. George R. Magee, Yerington, Nevada, Symptomatology, Clinical Diagnosis and Therapy.
Dr. David H. Lawrence and J. L. Terrell, Austin State School, Austin, Texas, Symptomatology of Relapsing Fever Superimposed on Neurosyphilis.
Dr. Sidney W. Bohls and Vernal Irons, Sc.D., Texas State Board of Health, Austin, Laboratory Diagnosis.
Vernon T. Schuhardt, M.A., associate professor of biology, University of Texas at Austin, Serology.
Dr. Hardy A. Kemp, dean, Ohio State University College of Medicine, Columbus, Dr. Fisher and Howard L. Evans, Dallas, Texas, Pathology and Immunity.
Dr. Bohls, Public Health Aspects.

The meeting of the American Association for the Advancement of Science will open in Dallas, December 29, to continue through January 3.

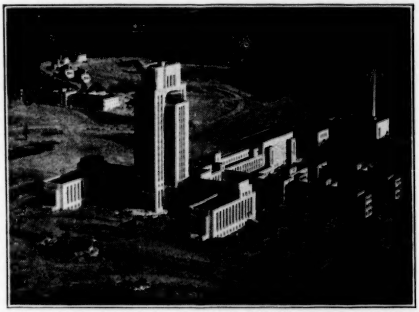
Government Services

First Aid Training by Bureau of Mines

The U. S. Bureau of Mines, Washington, D. C., during the last fiscal year gave first aid training to nearly 100,000 persons, raising the total number of persons who have received first aid certificates from the bureau of mines to more than 1,375,000. The bureau has also given instruction in the use of gas masks and in methods of handling explosions and fires in mines to about 75,000 persons. Nevertheless, in coal mines during the year there were seven major disasters with a total of 155 deaths, as compared with three major disasters and 206 deaths for the fiscal year 1940. To help reduce injuries in coal mines, the Congress of the United States passed on May 7 the Federal Mine Inspection Act, which empowers the Bureau of Mines to make inspections and investigations in coal mines. A corps of mine inspectors will be selected and laboratory facilities expanded to handle the work of investigating health and safety in coal mines.

New Naval Medical Center

The new U. S. Naval Medical Center at Bethesda, Md., which is now ready for occupancy, has been completed at a cost of $7,000,000. It is expected that moving from present facilities in Washington, D. C., will take place after January 1. The center includes the Naval Hospital, Naval Medical School, Naval Dispensary and Naval Dental School, situated on a 247 acre reservation. The tower of the administration building, which is set on a hill facing the Rockville Pike, will be devoted to wards and individual sickrooms, is twenty stories high and rises to a height of 270 feet above the Rockville Pike. The



The new U. S. Naval Medical Center.

fourth floor of the administration building extends the full length between the wings and will house principally the x-ray department and an auxiliary surgical service. The Medical Center consists of a central group of buildings, which includes administration offices, laboratories, classrooms, a surgical pavilion, two ward buildings, ship's service, commissary and recreation facilities, including a movie theater seating six hundred. The hospital corps quarters accommodate two hundred and eighty-five men, and the nurses' quarters accommodate seventy nurses. The hospital, which has a capacity of four hundred and fifty beds, in addition to an outpatient department, will utilize the clinical laboratories of the medical school. The Naval Medical School will conduct post graduate courses in phases of medicine peculiar to naval service, such as field hygiene related to service with the marines, submarine hygiene and physiologic problems related to deep sea diving, aviation medicine and tropical diseases. The training courses conducted for hospital corpsmen of the navy will include those for x-ray, chemistry, pharmacy, clinical laboratory, epidemiologic and blood plasma technicians. The Naval Medical Center will have a medical library large enough to accommodate seventy thousand volumes and a "crew's" library for the general use and recreation of patients and staff. Special facilities have been provided for research. Special attention will be given to epidemiology and to the effects of increased and decreased air pressure having a relation to the depth to which divers may descend and the heights to which aviators may climb.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Oct. 25, 1941.

National Health Good After Two Years of War

In previous letters the satisfactory condition of national health, which has actually improved under war conditions, was stated. These reports are now confirmed by Sir Wilson Jameson, chief medical officer of the Ministry of Health, who says that at the close of the second summer of war we have every reason to be thankful for the health of the country and that there is no reason to be pessimistic about the third winter, but that every conceivable care should be exercised and local authorities and the medical profession must remain alert. Most of the infectious diseases have declined steadily. Scarlet fever and diphtheria are less prevalent than a year ago. In the first week of September the number of cases of measles was only one sixth of the number in the corresponding week of last year. Cerebrospinal fever touched its lowest level for many months in the last week of August. There are still over 3,000 cases of whooping cough a week, but not long ago the number was well over 4,000. Enteric fever, notably paratyphoid, is more prevalent than in 1940. The ministry is pushing forward with diphtheria immunization. The percentage of children immunized in different cities varies from 60 upward and in one city has reached 95. Jameson points out that it costs 15 cents to immunize a child but over $150 to treat one for diphtheria in a hospital. There has been a rise in deaths from pulmonary tuberculosis, mostly among females of ages between 15 and 25 years. Deaths due to air raids have been least among children of school age, which is due to their evacuation to safer areas in the country. Possibly this also explains the decline of infectious diseases among them.

There is an epidemic of paratyphoid fever in Bristol, where a mobile team of the Harvard Medical Unit is working. Jameson says that if all who handle food would wash their hands before doing so these paratyphoid outbreaks would soon cease. One of the first persons to fall ill of the disease in Bristol was engaged in the preparation of food, and this was more or less responsible for the widespread outbreak. The Harvard team in Bristol consists of a medical officer, two laboratory workers, six hospital nurses and six public health nurses.

Medical Cooperation with Russia

A committee consisting of Lord Dawson, Sir Gowland Hopkins and Prof. J. A. Ryle has been formed to arrange the interchange between the British medical profession and that of the Soviet Union of the latest material on medicine. It is called the Anglo-Soviet Medical Committee. The members hold that such an interchange can be of value at present to members of the profession in both countries and that a large number of British physicians would be willing to cooperate in the work. Those interested and prepared to assist by abstracting English articles and translations of Russian work are invited to write to the committee at the house of the Royal Society of Medicine.

American Hospital in England

In the south of England is an all American complete field hospital with nine wards, a fully equipped kitchen, a pathologic laboratory and accommodation for the medical and nursing staff, consisting entirely of American volunteers. The roofs, floors, walls, every bolt and screw and all the furniture and equipment have been shipped from America as a gift from the American Red Cross and Harvard University. The hospital is constructed of timber throughout and was delivered on the site in prefabricated sections ready for immediate assembly. It has been put together under the supervision of Mr. Gwyer, an American. It is estimated that, by employing thirty men with a knowledge of this system of construction, a hospital of this size can be assembled and equipped in less than twenty-five weeks. The hospital incorporates a mobile field unit. The director is Dr. John E. Gordon, professor of preventive medicine and epidemiology at Harvard University. The university has also supplied the whole of the medical staff.

Reduction in Civilian Air Raid Casualties

The Ministry of Home Security has published figures showing a great reduction in civilian casualties from air raids since these reached a maximum in September 1940. For August 1941 the figures were 169 killed and 136 injured and detained in hospitals. From Sept. 3, 1939 to July 31, 1940, a period before air raids on this country developed, the civilians killed numbered only 310. Then raids on a large scale began and the number killed increased considerably: in 1940 August 1,085, September 6,955, October 6,335, November 4,751, December 3,829: in 1941 January 1,539, February 793, March 4,298, April 6,068, May 5,394, June 399, July 501. The great decline since May coincides with raids on a much smaller scale, which can be explained by the attack on Russia.

BUDAPEST

(From Our Regular Correspondent)

Oct. 3, 1941.

New Law on Protection of the Magyar Race

On October 10 the new law on the protection of the Magyar race will enter into force. The law forbids marriage between Jews and non-Jews. When Christians marry and four or at least three of their grandparents are Gentiles, according to their best knowledge, no documentary evidence has to be produced at the marriage registry offices. But if only two grandparents are or were Christians, rigorous certification is demanded as to the applicants' descent. In cases of marriage in spite of the new marriage law by misleading statements the law empowers the misled party to take legal steps for the dissolution of the marriage. The law prescribes severe punishment for Jews who have sexual intercourse with native Magyar, non-Jewish woman. Henceforth persons wanting to marry must have a thorough medical examination, must be made by city medical officers of health in towns and cities and district physicians in the country. The examinations are made gratuitously, a certain fee for these being paid by the state.

The Remains of Primitive Men in the Aggtelek Cave

Aggtelek, a stalactitic cave in upper Hungary, attracted the attention of archaeologists about the middle of the nineteenth century when Christian Raisz began to examine its queer formations. In the course of his investigations, in one section of the cave which is cut into halves by the water of Acheron, in the stalactites he hit on a whole series of skeletons placed one above the other. He named this part a mausoleum. In the vicinity of the skeletons he found various kitchen utensils and decorative objects. The forms of the vases pointed to a refined taste, and the coloring and finishing to an elaborate sense of art. Raisz could not explain their presence in the cave except as a result of the Tartar invasion. His assumption was that when Batu Khan's barbarian hoards invaded the country a part of the population sought refuge in the cave. Later scientific excavations refuted Raisz's assumption. In 1876 Professor Nyáry discovered two other caves in the vicinity of the mausoleum. Fragments of tile, human skeletons, the remainders of cave dwelling bears and other animals, lances made from bear bones, spear tips, chisels, amulets, axes and saws made of fish

bones came to light. They disclosed that the cave of Aggtelek had been a human living place beginning from the stone age through the paleolith and neolith and iron age until historical times. The seeds of wheat, lentil, rye, barley and the remainders of fourteen animals corroborate the assumption that the inhabitants of the cave knew agriculture and the breeding of useful animals. Examination of the skeletons revealed that they originated from primative Magyars and by scientific comparison they could be strictly delineated from skeletons originated from the invading Mongolians and Tartars. Many of the skeletons are on exhibit at the Ethnographical Museum in Budapest.

Tolerance of Young Cats for Chloroform

Katona and Besenyei studied chloroform narcosis on young cats of different ages. They observed that the blood pressure of grown up animals falls at the outset of narcosis, while that of young animals during the course of narcosis does not change much. In young animals periodic fluctuation of blood pressure is frequent in conjunction with partial respiratory paralysis about the end of narcosis. This phenomenon was observed by them also on grown up animals. The pulse rate of adult animals gradually decreases from the beginning of narcosis, while that of young animals decreases only after a transitory rise, and in deep narcosis it rises again. The respiration of adult animals is accelerated during the whole course of the narcosis; young animals breathe rapidly, but in deep narcosis the rate of respiration falls below normal. Partial respiratory paralysis with periodic respiration is of frequent occurrence: this is never observed on adult animals. The cause of death of adult animals from narcosis frequently is syncope. Katona and Besenyei believe that for the purposes of surgical narcosis chloroform is safer in the developing age than in adults but that does not mean it is so toxic as to justify its disuse in cases in which ether is contraindicated.

Formula for Estimation of Increase of Length and Weight of Infants

Dr. Joseph Lukács has compiled formulas with which one can easily calculate normal length and weight data. 1. The normal body weight of an infant is obtained by multiplying the age in months by 600 and adding this sum to its birth weight. If the latter is unknown, an average of 3,300 Gm. is taken. For instance, a 5 months old infant with a birth weight of 3,100 Gm. should weigh $5 \times 600 + 3{,}100 = 6{,}100$ Gm. The body weight of children between 1 and 12 years of age is obtained by subtracting 1 from the number of years, multiplying the remainder by 2 and adding 10. For instance, the weight of a child of 9 years is $9 - 1 = 8 \times 2 + 10 = 26$ Kg. These formulas give average figures. The weight of girls is slightly less than that of boys, so that if the formula is applied for girls the value obtained should be taken as optimal, while for boys it is minimal. 2. The normal height of an infant is obtained by multiplying its age in months by 2 and adding 52 to the sum obtained. Thus the length of an 8 months old infant is $8 \times 2 + 52 = 68$ cm. The height of a child of 2 to 4 years is obtained by multiplying the age in years by 10 and adding 60 to the sum. For instance, the normal height of a 3 year old child is $3 \times 10 + 60 = 90$ cm. The height of children between 5 and 14 is reckoned by multiplying the age in years by 5 and adding 80 to the sum. For instance, the height of a child of 6 is $6 \times 5 + 80 = 110$. In regard to the body length, the difference between boys and girls is negligible.

New Institution for Research on Nutrition

The Ministry of Internal Affairs has established a new institute for research in proper feeding. The institute, which is in Budapest, will be opened in the next few days. The building and equipment are modern and will accommodate two hundred country doctors, who will attend courses in the science of feeding. The staff of the institute comprises clinicians, chemical experts, economists and financial experts.

RIO DE JANEIRO

(From Our Regular Correspondent)

Oct. 11, 1941.

Brazilian and American Congress of Surgery

The third Brazilian and American Congress of Surgery, organized by the Brazilian College of Surgeons, will be held November 16-20 at Rio de Janeiro. The official subjects will be "Surgery of Pain," "Burns" and "Amputations from the Functional Point of View." This congress will draw to Rio de Janeiro many important surgeons from North and South America. Among the foreign surgeons will be Prof. Julio Diez of Buenos Aires, Argentina, and Prof. Manuel Riveros of Assuncion, Paraguay, who are official translators for the congress. The president will be Prof. Benedito Montenegro of the University of São Paulo.

Pulmonary Schistosomiasis (Mansoni)

A clinical study of pulmonary schistosomiasis (mansoni) has been made by Dr. João Alves Meira, assistant of the Faculty of Medicine of São Paulo and a specialist on tropical diseases. The 4 cases described show that there is a pulmonary form of schistosomiasis (mansoni). In some cases the diagnosis can be made only by examination of the sputum, by roentgenograms and by a therapeutic test. The pulmonary forms occur especially in patients who have visceral schistosomiasis of advanced degree.

The roentgen evidence of the pulmonary forms alone cannot be accepted, for confirmation by other data, such as the presence of the parasite's eggs in the sputum, is required. Eosinophilia may be transitory, but its presence is important.

The prognosis varies with the clinical form of the disease. The most serious manifestations are those caused by endarteritis complicated by cardiac insufficiency. When pulmonary aspects occur in advanced cases of the disease with a picture of hepatic cirrhosis and splenomegaly, the prognosis is generally grave.

Marriages

KENNETH L. MCGUIRE, Richland, Iowa, to Miss Alta Patricia McDEVITT of Spokane, Wash., in Fairfield, Iowa, October 9.

WILLIAM BEDWELL HARRELL JR., Little Rock, Ark., to Miss Erin Johnson Wallace of Birmingham, Ala., in October.

WALTER JOSEPH DUKSA, Hartford, Conn., to Miss Anna Emily Lennon of Washington, D. C., September 20.

FRANK TRESSLER LINZ to Miss Josephine Gwaltney McGowan, both of Tampa, Fla., October 24.

WILLIAM ALFRED MITCHELL, Newport News, Va., to Miss Ella Walker Hill in Roanoke, November 8.

CORNELIUS B. MURPHY, Alton, Iowa, to Miss Ethel Koelzer of LeMars, at Jordan, Minn., September 15.

WILLIAM PLEAS STONER, Memphis, Tenn., to Miss Edna May Wilson of Guston, Ky., in October.

OLIVE ANGA LUNDGREN, Minneapolis, to Mr. James Gordon Bateman of Great Falls, Mont., June 24.

JOSEPH ARCHER RAVENEL, Charleston, S. C., to Miss Mary Castrinos of New Orleans, November 5.

WILLIAM FRANCIS JACOBS to Miss Mary Margaret Haskins, both of Chicago, in October.

WILLIAM HIGGINSON to Miss Louise W. Sona, both of Nashville, Tenn., in October.

HANNS C. SCHWYZER, St. Paul, to Miss Margaret Nelsen of Minneapolis, October 18.

DERBERT MERENBLOOM, Cumberland, Ky., to Miss Nancy Ettin of Harlan in October.

JOHN THOMAS AKIN JR. to Miss Ladye Jane Akin, both of Atlanta, Ga., October 11.

JOSEPH E. PISETSKY, Brooklyn, to Miss Lillian Sax of New York, August 17.

HARRY GIBEL to Miss Pearl Charms, both of Brooklyn, October 19.

Deaths

Kenneth Daniel Blackfan ✠ Boston; Albany (N. Y.) Medical College, 1905; Thomas Morgan Rotch professor of pediatrics at Harvard Medical School and the graduate school; professor of pediatrics at the University of Cincinnati College of Medicine from 1920 to 1923; instructor in pediatrics from 1913 to 1917, associate from 1917 to 1919, and associate professor at Johns Hopkins University School of Medicine, Baltimore, 1919-1920, and lecturer in clinical pediatrics, 1920-1921; assistant at Washington University School of Medicine, St. Louis, 1911-1912; member and past president of the American Pediatric Society; member of the American Academy of Pediatrics, the New England Pediatric Society, Association of American Physicians and the American Society for Clinical Investigation; member of the Harvard Infantile Paralysis Commission; delegate, White House Conference on Child Health and Protection in 1930 and editor of the report in 1932; general secretary of the Fifth International Commission on Nutrition sponsored by the League of Nations in Berlin in 1932; was general secretary of the International Congress of Pediatrics; chairman of the General Advisory Committee on Maternal and Child Welfare under the Social Security Act, from 1935 to 1937; consultant of the Children's Bureau since 1935; member of the Commission on Graduate Medical Education since 1937; honorary member of the Philadelphia Pediatric Society; corresponding member of the Royal Society of Medicine of England (section of the study of disease in children) and the British Pediatric Association; member of the Council on Pharmacy and Chemistry of the American Medical Association from 1934 to 1935; member of the board of directors of the Infants' Hospital; aged 58; died, November 29, at Louisville, Ky., of a malignant tumor of the brain.

Frederick Taylor Lord ✠ Boston; Harvard Medical School, Boston, 1900; assistant in clinical medicine from 1905 to 1909, instructor from 1909 to 1930, clinical professor of medicine from 1930 to 1935 at his alma mater and since 1935 clinical professor of medicine emeritus; past president of the American Association for Thoracic Surgery; formerly vice president of the National Tuberculosis Association; president of the Massachusetts Tuberculosis League; member of the Association of American Physicians; fellow of the American College of Physicians; member of the American Society for Clinical Investigation and the American Clinical and Climatological Association; in 1917 member of the American Red Cross Commission to Serbia; served as visiting physician from 1912 to 1935 and member of the board of consultation since 1935 and in various other capacities at the Massachusetts General Hospital; served as a member of the advisory committees of the state department of public health and the city health department; member of the Boston Council of Social Agencies and chairman of the pneumonia and tuberculosis committees, Boston Health League; author of "Diseases of the Bronchi, Lungs and Pleura" and "Pneumonia"; co-author, with Dr. Roderick Heffron, of "Lobar Pneumonia and Serum Therapy," and "Pneumonia and Serum Therapy" and co-author, with Dr. Elliott S. Robinson and Dr. Heffron, of "Chemotherapy and Serum Therapy of Pneumonia"; aged 66; died, November 4, in the Baker Memorial of the Massachusetts General Hospital of subarachnoid hemorrhage.

James Porter Fiske, New York; College of Physicians and Surgeons, medical department of Columbia College, New York, 1891; surgeon, Charity Hospital and New York Maternity Hospital, 1891-1892; obstetric surgeon, Church Hospital, and attending surgeon, St. Elizabeth's Hospital, since 1893; orthopedic surgeon, New York Post-Graduate Hospital, from 1895 to 1899; orthopedic surgeon, Roosevelt Hospital, from 1893 to 1901; assistant surgeon, Bellevue Hospital, 1899-1900; orthopedic surgeon, New York Hospital; a founder of the schools for crippled children; past president of the Guild for Crippled Children of the Poor of New York; attending surgeon, Free School for Crippled Children from 1901 to 1913; charter member of the W. H. Davis School for Crippled Children; manager of the New York State Hospital for Crippled and Deformed Children; served during the World War; in 1924 was made a lieutenant colonel in the medical officers reserve corps; aged 74; was killed, October 25, in an automobile accident, near Commerce, Okla.

Menas Sarkas Gregory ✠ New York; Albany (N. Y.) Medical College, New York, 1898; professor of psychiatry, University and Bellevue Hospital Medical College, now known as the New York University College of Medicine, from 1922 to 1936 and since 1936 emeritus professor; formerly professor of psychiatry, New York Post-Graduate Medical School; member of the American Neurological Association, American Psychiatric Association and the Association for Research in Nervous and Mental Diseases; director of the psychiatric division, Bellevue Hospital, from 1904 to 1934 and since 1936 consultant; served during the World War; aged 69; died, November 2, while playing golf at Tuckahoe, N. Y.

Jack Schluger, Dayton, Ohio; Long Island College of Medicine, Brooklyn, 1936; was commissioned as a first lieutenant, medical corps reserve on June 2, 1936; served as a Civilian Conservation Corps camp surgeon in the Third Corps Area from June 15, 1936 to Jan. 4, 1937, and Oct. 25, 1938 to Sept. 30, 1939; was ordered to extended active duty with the United States Army, effective Aug. 1, 1941 and assigned to Kelly Field, Texas; on the staff of the Veterans Administration Facility, National Military Home; aged 32; was killed, October 14, in an airplane accident ten miles from Greensburg, Pa.

Grant Fletcher Mollring ✠ assistant surgeon, lieutenant, junior grade, United States Navy, Great Lakes, Ill.; University of Nebraska College of Medicine, Omaha, 1936; was appointed assistant surgeon in the medical corps of the United States Navy on Oct. 25, 1940; formerly served with the 10th Provisional Company of the United States Marine Corps at Quantico, Va., and also for a time at one of the new Atlantic bases; at one time surgical assistant at the Parkview Hospital, Rocky Mount, N. C.; aged 27; died, October 27, at the United States Naval Hospital, of acute anterior poliomyelitis.

Cary Robert Pollock, Denver; Vanderbilt University School of Medicine, Nashville, Tenn., 1915; member of the Colorado State Medical Society; was assigned to active duty Oct. 14, 1916 as a first lieutenant medical reserve for a course of instruction at the Army Medical School, Washington, D. C.; commissioned first lieutenant in the regular army medical corps March 28, 1917; promoted to major, March 28, 1918; resigned from the service Jan. 31, 1927; fellow of the American College of Surgeons; on the staff of the Presbyterian Hospital; aged 53; died, October 25.

Seth Selig ✠ New York; Columbia University College of Physicians and Surgeons, New York, 1921; member of the American Academy of Orthopedic Surgeons; fellow of the American College of Surgeons; medical officer of the city fire department; attending orthopedic surgeon to the Mount Sinai Hospital and the Montefiore Hospital for Chronic Diseases; consulting orthopedist to the Hillside Hospital, Blythedale Convalescent Home and the New York Jewish Guild for the Blind; aged 44; died, November 2, of coronary sclerosis.

Melville Black ✠ Denver; Bellevue Hospital Medical College, New York, 1889; professor of ophthalmology emeritus at the University of Colorado School of Medicine; member of the American Academy of Ophthalmology and Otolaryngology; fellow of the American College of Surgeons; past president and secretary of the Colorado State Medical Society; ophthalmologist, St. Luke's Hospital, Children's Hospital and the Colorado General Hospital; aged 75; died, November 12, in St. Joseph's Hospital of bronchopneumonia and pyelonephritis.

Max Talmey, New York; Ludwig-Maximilians-Universität Medizinische Fakultät, München, Bavaria, Germany, 1894; member of the Medical Society of the State of New York; chief ophthalmologist and aurist at the Yorkville Hospital, now the Beth David Hospital from 1902 to 1908; surgeon at Harlem Eye, Ear and Throat Infirmary from 1905 to 1917; author of a book entitled "The Relativity Theory Simplified"; aged 74; died, November 6, of chronic myocarditis.

George Ambrus ✠ New York; Magyar Királyi Ferencz József Tudományegyetem Orvostudományi Kara, Szeged, Hungary, 1922; formerly assistant professor of pathology at the University of Hungary; director of the New York Physicians and Diagnostic Laboratory; aged 45; died, November 14, in the New York Post Graduate Hospital of carcinoma of the jejunum and abdominal metastasis.

William N. Beggs, Denver; St. Louis Medical College, 1886; member of the Colorado State Medical Society; medical director of Sands Home, Edgewater; on the staff of the Jewish Consumptive Relief Society Sanatorium; aged 79; emeritus member of the staff of the Swedish National Sanatorium, Englewood, Colo., where he died, October 31, of myocarditis and arteriosclerosis.

William Kay Bartlett ✠ Colonel, United States Army, retired, San Francisco; Medico-Chirurgical College of Philadelphia, 1903; entered the medical corps of the United States Army as an assistant surgeon in 1907; served during the World

War; was promoted to the grade of colonel in 1933 and retired in 1935 for disability in line of duty; aged 63; died, November 2.

Thomas Francis Birmingham ✠ Galesburg, Ill.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1904; member of the American Urological Association; president-elect of the Knox County Medical Society; aged 58; on the staffs of the Galesburg Cottage Hospital and St. Mary's Hospital, where he died, November 1, of cirrhosis of the liver.

Guy Payne Sanderson, Vicksburg, Miss.; Vanderbilt University School of Medicine, Nashville, Tenn., 1923; member of the Mississippi State Medical Association and the Southeastern Surgical Congress; served during the World War; served the Vicksburg Hospital in various capacities; aged 43; died, October 30, of acute coronary occlusion.

Willard Richardson Pierce, Milford, Del.; Hahnemann Medical College and Hospital of Philadelphia, 1909; served during the World War; formerly member of the state legislature; member of the state board of health; on the staff of the Milford Memorial Hospital; aged 55; died, October 30, of myocardial degeneration.

Marion Albert Andreen ✠ Chicago; Northwestern University Medical School, Chicago, 1922; associate in otolaryngology at his alma mater; member of the American Board of Otolaryngology; aged 47; died, November 10, at the South Shore Hospital of carcinoma of the gallbladder and cerebral thrombosis.

Chauncey De Witt Beebe ✠ Sparta, Wis.; Northwestern University Medical School, Chicago, 1918; was president of the Seventh District Medical Society; for many years president of the school board of Sparta; on the staff of St. Mary's Hospital; aged 49; died, November 5, of heart disease.

James David McCord, Lynchburg, Tenn.; University of Nashville (Tenn.) Medical Department, 1909; member of the Tennessee State Medical Association; county health officer; medical director and owner of a hospital bearing his name; aged 59; died, October 31, of coronary thrombosis.

Homer Daniel McGill, Clarksburg, Tenn.; Nashville (Tenn.) Medical College, 1878; member of the Tennessee State Medical Association; past president of the Carroll County Medical Society; aged 84; died, October 30, of uremia and prostatic obstruction.

Wallace Henry Carver, Los Angeles; Colorado School of Medicine, Boulder, 1902; served during the World War; aged 66; died, October 5, in the Veterans Administration Facility, West Los Angeles, of bronchopneumonia, arteriosclerosis and nephrosclerosis.

Thomas Hudson Harter, Pasadena, Calif.; University of Pittsburgh School of Medicine, 1914; member of the California Medical Association; served during the World War; chief surgeon, City of Pasadena Emergency Hospital; aged 51; died, October 20.

Emery Wells Goembel ✠ Rockford, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1908; aged 60; on the staff of St. Anthony's Hospital, where he died, November 1, of coronary thrombosis.

Lucia Florence Vickery, Boston; Woman's Medical College of the New York Infirmary for Women and Children, New York, 1892; member of the Massachusetts Medical Society; aged 81; died, October 23, of carcinoma of the pancreas.

Benjamin B. Tout, Archie, Mo.; Columbian Medical College, Kansas City, 1901; member of the Missouri State Medical Association; formerly state senator; bank president; at one time postmaster; aged 74; died, October 29, of paralysis agitans.

Adolf M. Silten, New York; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, 1901; member of the Medical Society of the State of New York; on the staff of the Beth David Hospital; aged 63; died, November 3.

Edward Vincent Murphy, Montreal, Que., Canada; McGill University Faculty of Medicine, Montreal, 1914; formerly medical superintendent of the Alexandra Hospital; aged 56; died, October 26, in the Royal Victoria Hospital.

Atherton Perry Mason, Fitchburg, Mass.; Harvard Medical School, Boston, 1882; member of the Massachusetts Medical Society; formerly city bacteriologist; at one time member of the board of health; aged 85; died, October 20.

Herman Henry Moore, Markham, Va.; University of Toronto Faculty of Medicine, Toronto, Ont., Canada, 1908; Detroit College of Medicine, 1909; fellow of the American College of Surgeons; aged 61; died, October 20.

Charles J. Forney, Woodward, Okla.; Keokuk (Iowa) Medical College, College of Physicians and Surgeons, 1901; member of the Oklahoma State Medical Association; aged 71; died, October 23, in Shattuck of arteriosclerosis.

Blanche Horner Muldoon Bach, Pico, Calif.; Indiana University School of Medicine, Indianapolis, 1910; member of the Medical Society of the State of Pennsylvania; at one time school physician; aged 53; died, September 20.

Elbert Stevenson Dupuy ✠ Beckley, W. Va.; Miami Medical College, Cincinnati, 1901; formerly secretary of the Raleigh County Medical Society; for many years on the staff of the Beckley Hospital; aged 65; died, October 16.

Hugh Boleyn Sprague ✠ Salt Lake City, Utah; Jefferson Medical College of Philadelphia, 1905; served during the World War; formerly police surgeon and assistant city health commissioner; aged 63; died, October 11.

William Rufus King Beck, Jackson, Miss.; Birmingham Medical College, 1914; member of the Mississippi State Medical Association; served during the World War; aged 56; died, November 15, of coronary thrombosis.

William Roderick Dunbar, Truro, N. S., Canada; McGill University Faculty of Medicine, Montreal, Que., 1897; past president of the Nova Scotia Medical Society; aged 71; died, September 12, in New Glasgow.

Walter Oscar Bartlett ✠ Boston; Harvard Medical School, Boston, 1902; aged 62; died, November 5, in the Baker Memorial of the Massachusetts General Hospital of embolism of the right internal carotid artery.

Stonewall Jackson Emory, Navasota, Texas; Medical Department of Tulane University of Louisiana, New Orleans, 1893; past president of the Grimes County Medical Society; aged 69; died, October 17.

John W. Bolen, Galax, Va.; Medical College of Virginia, Richmond, 1885; member of the Medical Society of Virginia; aged 80; died, November 2, in St. Albans Sanatorium, Radford, of coronary sclerosis.

Louis Charles Barrette, Sacramento, Calif.; Washington University School of Medicine, St. Louis, 1925; member of the California Medical Association; aged 43; died, November 15, of malignant hypertension.

Margaret Coy-Bryan, Columbus, Ohio; Woman's Medical College of Pennsylvania, Philadelphia, 1934; member of the Ohio State Medical Association; aged 31; died, October 11, in the White Cross Hospital.

Charles Chessher Foster ✠ Granger, Texas; Medical Department of Tulane University of Louisiana, New Orleans, 1893; past president of the Williamson County Medical Society; aged 72; died, October 3.

Frederick Clarence Clarke, Bridgetown, Barbados, British West Indies; McGill University Faculty of Medicine, Montreal, Que., 1908; served during the World War; aged 58; died, September 20.

Chester Cole Ames ✠ Detroit; Detroit College of Medicine and Surgery, 1926; on the staff of the Receiving Hospital, Parkside Hospital and the Trinity Hospital; aged 43; died, November 2.

Frederick Cornelius Macdonald, Boston; Tufts College Medical School, Boston, 1901; member of the Massachusetts Medical Society; aged 65; died, October 27, of coronary thrombosis.

Edward Oliver Bonsteel, Cleveland; Cleveland Homeopathic Medical College, 1903; served during the World War; aged 73; died, November 5, of ruptured aneurysm of the abdominal aorta.

Goldburn H. Wilson, St. Louis; St. Louis College of Physicians and Surgeons, 1889; formerly member of the state legislature; aged 76; died, October 31, of coronary sclerosis.

Emile Roy, Tulsa, Okla.; Queen's University Faculty of Medicine, Kingston, Ont., Canada, 1901; member of the Oklahoma State Medical Association; aged 78; died, October 13.

Lemuel Martin Roberts, Little Falls, Minn.; Hahnemann Medical College of Philadelphia, 1883; member of the Minnesota State Medical Association; aged 79; died, October 9.

Olaf Magneson Sattre, Rice Lake, Wis.; Rush Medical College, Chicago, 1888; member of the State Medical Society of Wisconsin; bank president; aged 78; died, October 27.

James H. Guinn, Wilmington, Calif.; Kansas City (Mo.) Medical College, 1886; aged 85; died in July at Los Angeles of arteriosclerosis.

George E. Gwinn, San Antonio, Texas; Barnes Medical College, St. Louis, 1898; died in September.

Correspondence

INTERNATIONAL SOCIETY OF SURGERY

To the Editor:—The opportunity for gathering together a number of the fellows of the International Society of Surgery was presented by their attendance at the recent clinical congress of the American College of Surgeons in Boston, November 3-7.

The minutes of the meeting of certain American, Canadian and South American fellows of the International Society of Surgery at the Harvard Club of Boston, November 6, are as follows (forty-six fellows attended):

A report was given of the inaccessibility of the executive staff of the society following the collapse of France and Belgium. It was pointed out that the president, Dr. Mayer, the secretary general, Dr. Dejardin, and the treasurer, Dr. Lorthioir, were all Belgians and that we had not been able to get in touch with them since the occupation of Belgium.

It was pointed out that the National Committee for the United States and Canada, though it had no desire to continue in office, believed that the International Society of Surgery, the first and for nearly half a century the leading and most authorized exponent of surgical culture and achievement throughout the civilized world, was a necessary part of international medical relations and that temporarily the responsibility for continuing the existence of this society rested on the fellows in the Western Hemisphere.

In conformity with this feeling the committee presented certain resolutions for action by those present in order that the activities of the International Society of Surgery might be continued and fostered during the period of the present war.

The following resolutions were voted on and passed:

1. *Resolved*, That we, Fellows of the International Society of Surgery, here assembled in response to the call of the National Committee for the United States and Canada in Boston, Massachusetts, and coincidentally with the thirty-first Clinical Congress of the American College of Surgeons and in the absence, or lack, of all possible communication with the Executive Bureau at Brussels, do name, elect, and authorize the National Committee of the American Section, consisting of Elliott C. Cutler, M.D., Boston, Massachusetts, Rudolph Matas, M.D., New Orleans, Louisiana, and Eugene H. Pool, M.D., New York, New York, to take appropriate steps to continue their duties and functions until such time as the restoration of peace and order in Europe, and particularly in Belgium, will permit the International Society of Surgery to resume its normal prewar mode of existence.

2. *Resolved*, That the National Committee of the International Society of Surgery in the United States be likewise authorized to enlarge its own committee membership in any way that it may deem necessary to meet the requirements of the Committee and of the Society.

3. *Resolved*, That the National Committee shall be authorized by consultation and correspondence with Western Hemiphere colleagues to perfect further organization.

It was clear that some of the fellows did not feel that the American group had the authority to take over the executive duties of the central organization but felt that it was desirable for all fellows in the Western Hemisphere to join together in continuing the society by an enlargement of the present National Committee for the United States and Canada and by adding to the roster suitable members from all parts of the Western Hemisphere after consultation with the surgical academies and societies now existent. In particular the fellows from Venezuela, Argentina, Brazil and Cuba voiced the opinion that they and their colleagues from Central and South America would be glad to be consulted and to further this activity.

NATIONAL COMMITTEE FOR THE
UNITED STATES AND CANADA.

ELLIOTT C. CUTLER, M.D., Boston, Chairman.
RUDOLPH MATAS, M.D., New Orleans.
EUGENE H. POOL, M.D., New York.

POTENCY OF DIGITALIS PREPARATIONS

To the Editor:—In THE JOURNAL, October 11, page 1243, appeared an article calling attention to the increase in potency of the digitalis preparations in the U. S. Pharmacopeia XI, which became official June 1, 1936 (Bland, E. F., and White, Paul D.: The Strength of Digitalis in Clinical Use: A Warning). Unfortunately, not enough publicity was given to the change at the time when it took effect. Further, there has been some controversy as to just what the change was. This communication is written to present the intent of those responsible for the next revision of the U. S. Pharmacopeia.

The change in potency made at the time of the last revision was intended to bring the digitalis preparations of the U. S. Pharmacopeia XI into line with those of countries accepting the League of Nations Standards. In the U. S. Pharmacopeia XI (p. 136) appears the following statement regarding the unit in which the activity of digitalis preparations is to be expressed: "One United States Pharmacopeial Digitalis Unit is identical in potency with the International Digitalis Unit, as adopted in 1928 by the Permanent Commission on Biological Standardization of the Health Organization of the League of Nations. One International Digitalis Unit represents the activity of 0.1 Gm. of the International Standard Digitalis Powder." Powdered Digitalis of the U. S. Pharmacopeia XI is required to possess a potency of 10 U. S. P. units per gram and Tincture of Digitalis 1 U. S. P. unit per cubic centimeter. When these requirements were established it was generally believed that meeting them involved an increase in potency to 125 to 130 per cent of those official in the U. S. Pharmacopeia X (Edmunds, C. W.: The Potency of Digitalis Preparations of the 1936 Pharmacopeia, THE JOURNAL, July 22, 1939, p. 284).

The preparation of digitalis which was to serve as the new U. S. Pharmacopeia XI standard was prepared under the auspices of the Committee of Revision and then assayed against the 1926 International Standard Digitalis. When so examined it was found to be definitely stronger than the International Standard. When compared by the official one hour frog method in the laboratory of the chairman of the Subcommittee on Biological Assays, the late Dr. C. W. Edmunds, it was found that 0.745 Gm. of the United States Powder was equal in activity to 1.000 Gm. of the International Powder. This result did not create difficulty. It simply meant that 1 Gm. of the Pharmacopeial preparation Powdered Digitalis had to be equivalent in activity to 0.745 Gm. of the U. S. P. Reference Digitalis Powder instead of to 1.0 Gm.

However, there has not been universal acceptance of the correctness of this value of 0.745. Those who disagreed with Dr. Edmunds believed the Reference Digitalis Powder to be even stronger than he found it, which is another way of saying that his factor was too large. If this is true, then any digitalis preparation assayed against the U. S. P. XI Reference Digitalis Powder using this factor and meeting the Pharmacopeial requirement would actually be more than 130 per cent of the U. S. Pharmacopeia X strength. In the article by Bland and White which evoked this communication, the statement is made that "the majority of pharmacologists agree that by biological assay this increase in potency is approximately 25 to 30 per cent." I cannot speak for the majority of the pharmacologists, but most of the published biologic assay results indicate that the increase is something over 50 per cent, which brings the findings into line with those reported for clinical strength by Bland and White.

In 1939, because of a general feeling that the method of assay could be improved, a collaborative study was organized under the auspices of the U. S. P. XI of the assay of digitalis using frogs. At the request of the chairman of the U. S. P. XI Revision Committee, Prof. E. Fullerton Cook, and with the approval of Dr. Edmunds, chairman of the Subcommittee on Biological Assays, I have acted as chairman of this study. Gen-

erous collaboration has been received from university and manufacturing laboratories and the government control laboratories in both Canada and the United States. Studies by members of the group have confirmed the view that the present U. S. P. Reference Digitalis Powder is stronger than was intended.

Because of this fact, and also because of other characteristics which make the Reference Digitalis Powder unique, a new so-called "Study Powder" was prepared through the office of the chairman of the Revision Committee. This powder, a composite of assayed digitalis from a variety of sources, has now been extensively examined by several methods in a number of laboratories, and its characteristics are well known. The adoption of this powder as the standard of reference has been recommended to the Subcommittee on Biological Assays of the U. S. P. XII. The recommendation has been accepted, and when the powder has been adjusted to the potency of the International Standard, i. e., so that it contains 10 international units per gram, it is to become the Reference Digitalis Powder of the U. S. P. XII. The activity of all pharmacopeial digitalis preparations will continue to be expressed in terms of "Digitalis Units." One U. S. P. XII Digitalis Unit is to be the activity of 0.100 Gm. of this new Reference Digitalis Powder. Further, the comparison is to be made by the use of cats rather than frogs as the assay animal.

In the light of this explanation, qualification must be made of the statement by Drs. Bland and White, in footnote 6 of their paper, that in the twelfth revision of the U. S. Pharmacopeia the unit of potency has not been altered. The essential wording has perhaps not been seriously modified, although the U. S. P. XII will make it clear that the U. S. P. XII Reference Digitalis Powder is the reference standard, and that the published relationship to the International Standard is for purposes of information and not of definition. But the strength of the standard has been altered, and a new method of assay has been prescribed. These do in fact alter the unit.

The sole concern of the physician, however, is with the effect of these changes on the potency of official digitalis preparations. Practically all of the intensive studies of digitalis dosage were made prior to 1936, when the U. S. P. XI standards became effective. Most of the recent discussions have centered around the relationship of the U. S. P. XI digitalis potency to that of the digitalis in use prior to that time. How much stronger are present day preparations? What is to be the relative potency of the new preparations?

An answer to either of these questions presupposes a satisfactory knowledge of the strength of the earlier preparations. Exact information satisfactory to the biologic assayist is wanting on this point. However, there is adequate information on which to base a statement that should be satisfactory for those using digitalis in the clinic.

The U. S. P. XI Revision Committee intended to bring the official preparations of digitalis into conformity with international practice, which would have increased them to about 125-130 per cent of those previously official in the Tenth Revision. Actually those preparations which were adjusted to the new requirement have been found to be stronger than intended, running as high as 150 per cent. The U. S. P. XII preparations are to have the potency intended in the U. S. P. XI, namely about 125 per cent of those in use before 1936.

The implications of this statement should be emphasized. Official digitalis preparations of the U. S. P. XII will be somewhat weaker than those now current but will continue to be stronger than those of the period before 1936. This increase in potency must be recognized. The Revision Committee of the U. S. P. XII hopes that with the adoption of a new standard of reference, with a new method of standardization and with the greatly increased knowledge of biologic assays that has grown in part from attempts at solution of the controversial issues, this confusion may be cleared away. However, unless it is clearly understood that there has been an increase in the potency of digitalis preparations official since 1936, this confusion will continue.

Digitalis preparations official under the U. S. P. XI, effective since June 1, 1936, are stronger than those of the U. S. P. X. Adoption of the proposed U. S. P. XII Reference Digitalis Powder and definition of the U. S. P. XII Digitalis Unit as the activity of 0.100 Gm. of this powder will result in a continuation of this increase in potency. Digitalis preparations official in the U. S. P. XII will have a potency equal to about 125 per cent of those in use prior to 1936. Since most clinical studies of digitalis dosage on which present practices are based were made before 1936, it is important that the medical profession recognize that both current digitalis preparations and those to be official in the forthcoming Twelfth Revision of the U. S. Pharmacopeia are stronger than those of the U. S. Pharmacopeia X.

ERWIN E. NELSON, M.D.,
Department of Pharmacology, Tulane University of Louisiana School of Medicine, New Orleans.
Chairman, Subcommittee on Biological Assays, U. S. P. XII Revision Committee.

TRANSFUSION OF BLOOD AND BLOOD SUBSTITUTES

To the Editor:—The concluding paragraph of the editorial "Transfusion of Blood and Blood Substitutes" (THE JOURNAL, November 8, p. 1627) contains the statement "The dried plasma promises to be the eventual solution of the problem for war emergency because of the greater stability, smaller bulk and the relative simplicity of packing, storing and transporting." This seems to me to be an oversimplification of the problem. The experimental and clinical studies in Great Britain and in this country have proved conclusively the efficacy of transfusions of plasma and serum in the first aid treatment of shock. Even shock accompanying hemorrhage can be treated successfully by restitution of the blood volume with serum or plasma, provided the bleeding is controlled. The personnel of the British Army Blood Supply Depot under the command of Col. L. E. H. Whitby has had a rare opportunity to study the treatment of shock in air raid casualties (Kekwick, A.; Maycock, W. D; Marriott, H. L., and Whitby, L. E. H.: Diagnosis and Treatment of Secondary Shock: A Study of Twenty-Four Cases, *Lancet* **1**:99 [Jan. 25] 1941). They found that from 2 to 6 pints (1 to 3 liters) of citrated plasma was required for the adequate treatment of shock accompanying hemorrhage. But they expressed the opinion that 1 of every 3 pints of fluid administered should consist of whole blood when large amounts of plasma were required. This was advised to obviate the further dilution of the recipient's erythrocytes and is supported by the experience of Scudder and Self (Controlled Administration of Fluid in Surgery, *New England J. Med.* **225**:679 [Oct. 30] 1941).

But when death from shock has been averted by this treatment, the patient frequently faces the hazard of an urgent major surgical operation under general anesthesia. With the widespread use of blood transfusions in this country in recent years the opinion has been formed that patients with posthemorrhagic anemia tolerate operations much better if the oxygen carrying capacity of the blood is enhanced by the transfusion of erythrocytes. Blalock has stated that "every effort should be made to restore the red blood cell count and the blood volume to normal prior to operation" (Principles of Surgical Care, St. Louis, C. V. Mosby Company, 1940, p. 148). In large civilian general hospitals in this country about two thirds of all whole blood transfusions are administered to surgical patients. The indication for most of these transfusions is the correction of anemia before operation. Undoubtedly the same indication will prove

as frequent in military medicine. Plasma will not be suitable for this phase of the war emergency.

The recent development of blood substitutes for use in military medicine has resulted in much confusion for civilian practitioners. In warfare the ease of storage, transportation and administration of such products is a much more potent argument in their favor than it is in civilian practice, in which the facilities for laboratory procedures and refrigeration are adequate or easy to obtain. For the civilian hospital, methods are readily available for the manufacture from preserved whole blood of liquid plasma stored either at room temperature or in the refrigerator. The production and storage of frozen plasma is also feasible. Conclusive evidence has not been introduced to discredit any of these products.

In three places in the editorial are references to the fact that the use of plasma or serum obviates the necessity of typing and cross matching of bloods. Here again the argument which correctly carries great weight in military medicine has actually only slight application to civilian practice. Many physicians are impressed by the argument of the saving of time in an emergency because the laboratories on which they rely are employing time consuming methods for the typing and cross matching of blood. When the centrifugation technic of Landsteiner is used it is possible to type or cross match blood accurately in five minutes. By utilizing a blood bank in the University Hospitals of the State University of Iowa it has been possible to have whole blood running into the recipient's vein within fifteen minutes after the demand for a transfusion is made. During the interval mentioned, the recipient's blood group is determined and his serum and cells cross matched with those of an appropriate specimen in the refrigerator. Rabbit antiserum, now commercially available, indicates the group of the erythrocytes thirty seconds after it is mixed with the cell suspension. Preserved blood of group O can be administered to any recipient in an emergency without preliminary typing of the recipient or cross matching of the bloods. The addition of the recently developed A and B substances to the blood of group O neutralizes the agglutinins in the plasma and promises to obviate most of the objections to the employment of universal donor blood (Witebsky, Ernest; Klendshoj, N. C., and Swanson, Paul: Preparation and Transfusion of Safe Universal Blood, THE JOURNAL, June 14, p. 2654).

A consideration of civilian practices in the transfusion of plasma and whole blood is not entirely foreign to the war emergency. In this time of "total war," in which civilian casualties may be numerous, sources of readily available whole blood and plasma are extremely valuable to the community. They may serve as depots from which the military forces may draw. In times of peace there are myriad examples in which small community hospitals have suddenly been faced with the task of treating scores of victims of a railway wreck, an earthquake, a hotel fire or multiple automobile accidents. The presence of a blood and plasma bank may appreciably affect the mortality resulting from such disasters.

The development of methods by which whole blood can be preserved for thirty days in a refrigerator in a modified Rous-Turner dextrose-citrate mixture (DeGowin, E. L.; Harris, J. E., and Plass, E. D.: Studies on Preserved Human Blood: I. Various Factors Influencing Hemolysis, THE JOURNAL, March 9, p. 850) has made possible the operation of blood banks on a much smaller scale than is feasible when the time of preservation is shorter. At the expiration of the limit of storage the dilute dextrose-citrate plasma may be aspirated from the erythrocyte layer with a minimum of equipment and the plasma can be kept almost indefinitely in the refrigerator (Alsever, J. B., and Ainslie, R. B.: A New Method for the Preparation of Dilute Blood Plasma and the Operation of a Complete Transfusion Service, *New York State J. Med.* **41**:126 [Jan. 15] 1941). This dilute plasma has been shown to be efficacious in the treatment of shock (Besser, E. L.: Stored Dextrose-Citrate Plasma in the Treatment of Operative Shock, *Arch. Surg.* **43**:451 [Sept.] 1941). The establishment of blood and plasma banks in thousands of smaller hospitals in the country not only would make these institutions more valuable in the treatment of the emergencies that arise in civilian practice but would also build a great military reserve which could be utilized much as the blood banks in cities in Great Britain are now being used in the treatment of air raid casualties.

ELMER L. DEGOWIN, M.D., University Hospital, Iowa City.

HEPARIN FOR PROLONGED COAGULATION TIME

To the Editor:—The remarks of de Takats in THE JOURNAL (Oct. 18, 1941, p. 1378) on the unusually prolonged coagulation time in our case of cavernous sinus thrombophlebitis treated with heparin are well received. Since this was the first case of this kind which we were fortunate enough to be able to treat with heparin, we attempted to approximate the regimen which had proved so successful in Schall's hands (THE JOURNAL, Aug. 23, 1941, p. 581). We are aware from the experience of others and our own subsequent experience that in cases of uninfected thrombophlebitis a coagulation time approximating twenty minutes seems to be adequate. However, in cases of septic cavernous sinus thrombophlebitis, in which the mortality is admittedly high and the prognosis always grave, it seems to us that a more prolonged coagulation time provides a greater margin of safety. We agree with de Takats that the complication which we encountered was due to the extremely high levels of coagulation time (three and four hours) which we attained. In any future cases we would attempt to avoid these extreme levels but still maintain a coagulation time considerably above twenty minutes.

It was not our intention to deter others from the use of heparin in any case of thrombophlebitis. Rather, we had hoped to help stimulate its more general use in these cases, but with a clearer realization of the occurrence of a complication which may ensue.

It remains for the future and the experiences of many observers to establish the optimum level of the coagulation time in these cases of septic thrombophlebitis.

IRVING L. ERSHLER, M.D.
IRL H. BLAISDELL, M.D.
Syracuse, N. Y.

SKIN TEST FOR DIAGNOSIS OF PREGNANCY

To the Editor:—It has recently been called to my attention by a number of physicians that a new cutaneous test for the diagnosis of pregnancy based on the report of Falls, Freda and Cohen (*Am. J. Obst. & Gynec.* **41**:431 [March] 1941) is now being actively detailed by a commercial pharmaceutic organization as a "simplified, economical, reliable diagnostic skin test for pregnancy."

A recent report by Dr. Arnold F. Snyder and myself (*ibid.* **42**:738 [Oct.] 1941) lists one hundred and fifty such tests using the exact technic described by Falls and his co-workers, in which an accuracy of only 75 per cent could be attained.

A word of caution should be interposed at this point in order to warn the practitioner not to be hasty in placing too much confidence in this reaction until further reports in the literature can support the contention of Falls and his colleagues in the accuracy of their test. Not only has the test been unreliable in our hands but a number of other investigators using smaller series of cases have informed me of the inaccuracy of this test in many instances.

ABNER I. WEISMAN, M.D., New York.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE

CHICAGO, Feb. 16-17, 1942. Council on Medical Education and Hospitals, Sec., Dr. William D. Cutter, 535 North Dearborn Street, Chicago.

MEDICAL CORPS, UNITED STATES NAVY

Examination. Assistant Surgeon with the permanent rank of Lieutenant (junior grade) and Acting Assistant Surgeon with the probationary rank of Lieutenant (junior grade), Jan. 5-9. Examination will be held at the Naval Hospitals at Chelsea, Mass., Newport, R. I., Brooklyn, Philadelphia, Norfolk, Va., Charleston, S. C., Pensacola, Fla., Corpus Christi, Tex., San Diego and Mare Island, Calif., Puget Sound, Wash., Great Lakes, Ill., Pearl Harbor, T. H., and Naval Medical Center, Washington, D. C. Apply Bureau of Medicine and Surgery, Navy Department, Washington, D. C.

BOARDS OF MEDICAL EXAMINERS

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Examinations of boards of medical examiners and boards of examiners in the basic sciences were published in THE JOURNAL, December 6, page 2002.

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS: Parts I and II. Various centers, Feb. 9-11. Exec. Sec., Mr. Everett S. Elwood, 225 S. 15th St., Philadelphia.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: *Written. Part I.* Various centers, March 31. Final date for filing application is Dec. 31. Sec., Dr. Paul M. Wood, 745 Fifth Ave., New York City.

AMERICAN BOARD OF INTERNAL MEDICINE: *Oral.* April in advance of the meeting of the American College of Physicians and June, in advance of the meeting of the American Medical Association. Applications should be on file 6 weeks in advance of the date of oral examination. Sec., Dr. William S. Middleton, 1301 University Ave., Madison, Wis.

AMERICAN BOARD OF NEUROLOGICAL SURGERY: New York, June. Sec., Dr. R. Glen Spurling, 404 Brown Bldg., Louisville.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY: *Written. Part I. Group B.* Various centers, Jan. 3. *Oral. Part II. Groups A and B* Atlantic City, May or June. Final date for filing application is March 1. Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh.

AMERICAN BOARD OF OTOLARYNGOLOGY: *Oral and Written. All Groups.* Philadelphia, June, preceding the meeting of the American Medical Association. Final date for filing application is March 1. Sec., Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, Neb.

AMERICAN BOARD OF PATHOLOGY: St. Louis, March 30-31. Final date for filing application is Jan. 30. Sec., Dr. F. W. Hartman, Henry Ford Hospital, Detroit.

AMERICAN BOARD OF PEDIATRICS: *Oral.* Los Angeles, April 22, preceding the Region IV meeting of the American Academy of Pediatrics, Cleveland, May 13, preceding the Region III meeting of the American Academy of Pediatrics. *Written.* Locally, February 14. Sec., Dr. C. A. Aldrich, 707 Fullerton Ave., Chicago.

AMERICAN BOARD OF RADIOLOGY: *Oral. All Groups.* Atlantic City, June 4. Final date for filing application is April 1. Sec., Dr. Byrl R. Kirklin, 102-110 Second Ave., S. W., Rochester, Minn.

AMERICAN BOARD OF UROLOGY: *Written.* Various centers, December. Sec., Dr. Gilbert J. Thomas, 1009 Nicollet Ave., Minneapolis.

Illinois June Report

The Illinois Department of Registration and Education reports the written examination (graduates of foreign schools given also a practical test) for medical licensure held at Chicago, June 24-26, 1941. The examination covered 10 subjects and included 100 questions. An average of 75 per cent was required to pass. Three hundred and twenty-eight candidates were examined, 310 of whom passed and 18 failed. The following schools were represented:

School	PASSED	Year Grad.	Number Passed
Howard University College of Medicine	(1939), (1940)		2
Chicago Medical School	(1937), (1940), (1941, 33)		35
Loyola University School of Medicine	(1941, 46), (1941, 5)*		51
Northwestern University Medical School, (1940), (1941, 25)	(1941, 5)*		32
Rush Medical College	(1939, 3), (1940, 17), (1940, 2)*		22
Univ. of Chicago, The School of Medicine	(1940, 5), (1940, 3)*		8
University of Illinois College of Medicine, (1940), (1940),* (1941, 94)	(1941, 15)*		111
Harvard Medical School	(1935)		1
St. Louis University School of Medicine	(1940)		1
Univ. of Rochester School of Medicine and Dentistry	(1940)		1
University of Oregon Medical School	(1940)		1
Hahnemann Med. College and Hospital of Philadelphia	(1940)		1
Jefferson Medical College of Philadelphia	(1940)		1
University of Pennsylvania School of Medicine	(1940)		1
University of Toronto Faculty of Medicine	(1932), (1938)*		2
McGill University Faculty of Medicine	(1937)*		1
Medizinische Fakultät der Universität Wien (1906), (1920), (1924), (1924),* (1925), (1927), (1929), (1935, 2), (1936, 2), (1937)	(1938)		13
Universzita Komenského Fakulta Lekarska, Bratislava	(1938)		1
Albert-Ludwigs-Universität Medizinische Fakultät, Freiburg	(1920), (1922)		2
Eberhard-Karls-Universität Medizinische Fakultät, Tübingen	(1919)		1
Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin	(1922), (1923)		2
Hamburgische Universität Medizinische Fakultät	(1924)		1
Johann Wolfgang Goethe-Universität Medizinische Fakultät, Frankfurt-am-Main	(1932)		1
Kaiser-Wilhelms-Universität Medizinische Fakultät, Strassburg	(1905)		1
Rheinische Friedrich-Wilhelms-Universität Medizinische Fakultät, Bonn	(1915), (1920)		2
Schlesische-Friedrich-Wilhelms-Universität Medizinische Fakultät, Breslau	(1922)		1
National University of Athens School of Medicine	(1936)		1
Magyar Királyi Erzsébet Tudományegyetem Orvostudományi, Pecs	(1921), (1933)		2
Magyar Királyi Pázmány Petrus Tudományegyetem Orvosi Fakultasa, Budapest	(1939)		1
Regia Università degli Studi di Bologna. Facoltà di Medicina e Chirurgia	(1937)		1
Regia Università degli Studi di Milano. Facoltà di Medicina e Chirurgia	(1938)		1
Regia Università degli Studi di Roma. Facoltà di Medicina e Chirurgia	(1938)		1
Regia Università di Torino Facoltà di Medicina e Chirurgia	(1932)		1
Univ. Basel Medizinische Fakultät	(1936), (1939, 2), (1940)		4
Université de Lausanne Faculté de Médecine	(1939, 2)		2

School	FAILED	Year Grad.	Number Failed
University of Toronto Faculty of Medicine	(1938)		1
Medizinische Fakultät der Universität Wien, (1921), (1922), (1924)	(1935)		4
Deutsche Universität Medizinische Fakultät, Prag	(1932)		1
Friedrich-Wilhelms Universität Medizinische Fakultät, Berlin	(1925)		1
Hamburgische Universität Medizinische Fakultät	(1922)		1
Johann Wolfgang Goethe-Universität Medizinische Fakultät, Frankfurt-am-Main	(1920)		1
Julius-Maximilians Universität Medizinische Fakultät, Würzburg	(1923)		1
Universität Köln Medizinische Fakultät	(1922)		1
Magyar Királyi Erzsébet Tudományegyetem Orvostudományi, Pecs	(1923)		1
Regia Università degli Studi di Bologna. Facoltà di Medicina e Chirurgia	(1935), (1937)		2
Regia Universita degli Studi di Genova. Facoltà di Medicina e Chirurgia	(1939)		1
Universität Basel Medizinische Fakultät	(1939)		1
Universität Bern Medizinische Fakultät	(1934)		1
Université de Geneve Faculté de Médecine	(1938)		1

Twenty-six physicians were successful in the practical test for reciprocity and endorsement applicants held in Chicago, June 26. The following schools were represented:

School	PASSED	Year Grad.	Reciprocity with
University of Arkansas School of Medicine	(1940)		Arkansas
Northwestern University Medical School, (1940) Missouri	(1937)		Minnesota,
Rush Medical College	(1938)		Ohio
Indiana University School of Medicine	(1936), *(1939)		Indiana
University of Louisville School of Medicine	(1934)		Kentucky
St. Louis Univ. School of Medicine (1936), (1939), (1940, 3)			Missouri
Washington University School of Medicine. (1937), (1940, 3)			Missouri
University of Nebraska College of Medicine	(1940, 2)		Nebraska
Ohio State University College of Medicine	(1936), (1939)		Ohio
Jefferson Med. College of Philadelphia. (1930) Penna.,	(1931)		Minnesota
Meharry Medical College	(1938)		Tennessee
University of Wisconsin Medical School	(1938)		Kansas

School	PASSED	Year Grad.	Endorsement of
Northwestern University Medical School	(1941)		N.B.M.Ex.
McGill University Faculty of Medicine	(1935)		N.B.M.Ex.

* Licenses have not been issued.

Arizona October Report

The Arizona State Board of Medical Examiners reports the written examination for medical licensure held at Phoenix, Oct. 7-8, 1941. The examination covered 10 subjects and included 100 questions. An average of 75 per cent was required to pass. Eight candidates were examined, all of whom passed. Five physicians were licensed to practice medicine by reciprocity and 1 physician was so licensed on endorsement of credentials of the National Board of Medical Examiners. The following schools were represented:

School	PASSED	Year Grad.	Number Passed
College of Medical Evangelists	(1940)		1
University of Southern California School of Medicine	(1941, 2)		2
George Washington University School of Medicine	(1939)		1
Northwestern University Medical School	(1940)		1
Rush Medical College	(1939)		1
Tulane University of Louisiana School of Medicine	(1940)		1
University of Toronto Faculty of Medicine	(1940)		1

School	LICENSED BY RECIPROCITY	Year Grad.	Reciprocity with
College of Medical Evangelists	(1921)		California
Northwestern University Medical School	(1934)		Texas
Rush Medical College	(1938)		Colorado
University of Minnesota Medical School	(1915)		Minnesota
University of Nebraska College of Medicine	(1935)		Nebraska

School	LICENSED BY ENDORSEMENT	Year Grad.
Cornell University Medical College	(1938)	

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Liability of Medical Service Corporation for Negligence of Employees.—One of the defendants, a hospital association incorporated for profit, contracted with the Washington High School of Portland, Ore., "to provide medical and surgical and hospital services, . . . for the members of the football squad of the school." The plaintiff, a member of the school's football squad, injured his left shoulder and arm in games on Sept. 14 and Sept. 20, 1935. The day after the second game he visited the hospital association's offices and was given medical attention by one of the other defendants, Shields, a physician employee of the association. Shields made a diagnosis of an "acromioclavicular bruise," gave the boy analgesic balm to apply to the shoulder if he should ever again suffer a similar injury and advised him that he could continue to play football. Apparently the plaintiff had suffered a dislocation of the left shoulder and in playing in succeeding games suffered repeated dislocations, consulting employees of the association following several such injuries, but the condition remained undiagnosed. By the following August the shoulder and arm were in such a condition that surgery was the only available remedy. Eventually he brought an action for malpractice against the hospital association, two physician employees, Shields and Sisson, and a lay employee, Jones. The trial court sustained a motion for nonsuit as to the lay employee but denied it as to the other defendants. The jury found in favor of the plaintiff and, from judgment on the verdict, the hospital association and the physicians appealed to the Supreme Court of Oregon.

It was first contended that the defendant hospital association was not liable for any negligent acts performed by the defendant physicians because the physicians were independent contractors rather than employees of the association. The association, it appeared, was organized under the laws of Oregon to furnish medical, surgical and hospital services for profit. The defendant physicans were paid a monthly salary by the company and were furnished office space in the company offices. They were permitted to have their own patients but, in the offices furnished them, they rendered the professional services which the company agreed to furnish under its various contracts. Obviously, said the Supreme Court, under the circumstances, the relationship existing between the association and the physicians was that of master and servant and the association, by the doctrine of respondeat superior, is liable for the physicians' negligence. The association argued that the very nature of a physician's services precludes the physicians from being subject to the association's control and therefore makes the physicians independent contractors. While, answered the Court, in many instances the relationship between the parties may be tested by the right or lack of right to exercise control over an agent as to the manner and details of his work, in a case of this kind such a test cannot be determinative, since the test must give way to the rule that one cannot absolve oneself of contractual obligations by devolving the performance of those obligations on a stranger to the contract. If the hospital association has provided services in a negligent manner, it has not complied with its contractual obligations and it cannot secure immunity from liability for the negligence of the physicians it employed to perform its contractual obligations by pleading that it exercised due care in the selection of those physicians.

It was next contended that there was no evidence of malpractice against the association and the defendant Shields. The plaintiff testified that on September 21, the day after his second injury, he visited Shields in the office furnished by the defendant company and told him that in a football game on September 14, when a pile of players got off of him, it felt as though his shoulder had been broken because "the bone was sticking out, it felt like," that two of the officials of the game snapped it in some way and it seemed to jump back in place, and that a similar injury was sustained in the game on September 20. The plaintiff then testified that Shields examined his arm, diagnosed his condition as an acromioclavicular bruise, gave him some analgesic balm to use if the same injury occurred again and told him that he could continue to play football. A physician, called by the defendants, on the basis of a history of the case given to him in August 1936, testified that at that time he made a definite diagnosis of recurrent dislocation of the shoulder and recommended an operation as the only remedy for the condition then existing. While, said the Court, there was no evidence that, in his examination of the plaintiff, Shields failed to use the tests approved by standard medical practice, it does not, however, necessarily follow that he was not guilty of negligence. There was medical testimony from which the jury could have found that the plaintiff, in recounting his injuries to Shields, described, characteristically, a dislocation of his shoulder, and therefore, the jury could have found that a physician, exercising ordinary care and skill in making his diagnosis, should have discovered the true nature of the plaintiff's ailment. It was conceded that the proper treatment for a recent dislocation of the shoulder was immobilization of the shoulder and arm for a period of time and that advice to the patient to play football would be highly improper. If, therefore, as the plaintiff testified and Dr. Shields vigorously denied, such advice was given, the evidence was sufficient to take the case to the jury, and the court committed no error in denying the motions for nonsuit and directed verdict with respect to the hospital association and Shields.

With respect to another one of the physician defendants, Sisson, the Court held that there was no evidence tending to prove that any act on his part was the proximate cause of the plaintiff's injury. The plaintiff contended, however, that since he had sued the defendants jointly, if negligence was proved with respect to any defendant, Sisson was necessarily also liable. The Court held, however, that in this case, essentially, the corporate defendant, the hospital association, had been sued for tort and with it were joined as defendants the corporation's employees who were charged with acting on its behalf in the commission of the wrongful acts. An employee cannot be held liable for the acts of a co-employee, said the Court, if he in no way participated in the acts on which the liability is sought to be predicated. Consequently Sisson's liability turns altogether on what he himself did or failed to do and he could in no wise be held responsible for the negligence of Shields. The Court accordingly held that the case should be dismissed as to Sisson.

At the trial, a witness called by the plaintiff was permitted, over objection, to testify that it was the policy of the high school concerned not to allow a boy to play football unless the hospital association "said it was all right to use him." The admission of this evidence, the Court held, was erroneous. The contract, said the Supreme Court, between the hospital association and the high school contained no provision requiring the company to keep the school authorities advised as to the physical condition of all the members of the football squad or to instruct the high school officials not to permit a boy to play because of his physical condition. The effect of the objectionable testimony was to inject into the case an issue not justified by the terms of the contract and not tendered by the plaintiff's complaint. Without this issue the whole case of the plaintiff depended on his unsupported testimony that Dr. Shields had advised him to continue playing football. If the jury believed Dr. Shields as against the plaintiff, it must necessarily have found for the defendants, because a wrong diagnosis in itself creates no liability. Since the testimony was received one may presume that it was commented on in argument, and, since there was no claim on the part of the defendants that any of them had advised the school authorities to have the plaintiff discontinue his football activities, prejudice to the defendants' case in all probability resulted.

The judgment was accordingly reversed and the cause remanded to the trial court with directions to enter a judgment of nonsuit in favor of Sisson and, apparently, to proceed with a new trial with respect to the hospital association and Shields. —*Giusti v. C. H. Weston Co., 108 P. (2d) 1010 (Ore., 1941).*

Malpractice: Alleged Failure of Dentist to Curet After Extraction and Discovery of Necrosis of the Mandible.—Fred Specht consulted the defendant dentist, Jan. 29, 1940, relative to a "loose tooth." After an examination, but without taking roentgenograms, the dentist extracted the tooth and discovered that his patient was suffering with necrosis of the mandible. Just what treatment, if any, the dentist then undertook, the reported decision does not make clear. The patient became ill with "systemic infection or streptococcus from which he died on February 6, 1940." The patient's mother instituted an action for damages against the dentist, alleging that in caring for her son the dentist had failed to exercise the care required of him by law in that before the extraction the dentist failed to have a proper roentgenogram taken, that the dentist failed to use sanitary, clean and sterilized instruments in the probing and extracting of the tooth and its root, that although the dentist discovered after the extraction that the decedent had a necrosis of the mandible and was in great danger of infection he failed to send decedent to a hospital to have the cavity properly curetted and failed himself to curet "properly, sanitarily, and sufficiently," and that the dentist negligently allowed his patient to become ill with "systemic infection or streptococcus" from which he died. The trial court sustained a general demurrer to the complaint and the decedent's mother appealed to the court of appeals of Georgia, division 2.

The Georgia code, section 84-924, said the court, requires a person professing to practice medicine or surgery for compensation to bring to the exercise of his profession a reasonable degree of care and skill. Any injury resulting from a want of such care and skill is a tort for which a recovery may be had. These provisions of the code apply also to a licensed dentist in the practice of his profession. The standard prescribed by the code when applied to the facts and circumstances of any particular case must be taken and considered to be such a degree of care and skill as, under similar conditions and like surrounding circumstances, is ordinarily employed by the profession generally. Not only must a dentist possess the requisite care and skill but he must exercise these qualifications. A dentist is not an insurer or warrantor that the exercise of his professional judgment will effect a cure of the patient. Nor is he obliged to bring to the exercise of his profession the utmost skill. If he measures up to the qualifications and applies the reasonable care and skill legally required of him he is not responsible for a mistake of judgment. If, however, an error of judgment is so gross as to be inconsistent with that degree of care and skill which a dentist should possess and exercise, liability may result when an injury is produced. A dentist in practicing his profession is under duty not only to use the requisite care and skill in a particular operation but also to give such after-treatment to the patient as the necessities of the case demand, in the absence of any special agreement to the contrary. Applying the aforestated principles of law, continued the court, to the facts alleged in the present petition, that in extracting the root of a tooth of the decedent the dentist negligently failed to use sanitary instruments and, on discovering that the jawbone of the patient was diseased and in great danger of infection, negligently failed to curet the cavity or to send the patient to a hospital for such after-treatment, in consequence of which alleged negligence "systemic poisoning or streptococcus" developed in the jawbone, causing the death of the patient, a jury question was presented as to whether or not the dentist was guilty of such gross error of judgment as to amount to a failure to exercise the requisite care and skill toward the patient, and the trial court erred in sustaining the defendant's general demurrer to the petition. The court accordingly reversed the judgment of the trial court in favor of the dentist.—*Specht v. Gaines, 16 S. E. (2d) 507 (Ga., 1941).*

Malpractice: Death from Pulmonary Embolism Following Tonsillectomy and Turbinectomy.—The patient underwent an operation for the removal of his tonsils and the turbinated bones of his nose, the operation being performed by a physician employed by the patient. A hospital resident physician, an anesthetist and an instrument nurse assisted in the operation. About five hours after the completion of the operative procedures the patient died, death being attributed to "pulmonary embolism of the left upper lobe of the lung, with an infarct of the same upper lobe." Thereafter the widow and the son of the deceased patient brought suit against the hospital, the operating physician, the resident physician, the anesthetist and the instrument nurse. The trial court directed a verdict for the defendants and the plaintiffs appealed to the Court of Appeals of Maryland.

The operation was performed under a general anesthetic and an ether tube was used. The plaintiffs' evidence tended to show that this tube was left, unfastened, in the patient's mouth when he was returned to his room and that several hours later a friend visited the patient and found him unattended, "fighting for his breath" and waving "his hands in a frantic way." The patient died two hours later. The apparent contentions in this case were (1) that the defendants were negligent in permitting the ether tube to remain untied in the patient's mouth which "allowed the blood, serum and mucus to run down into the windpipe, clot, and stop the air from passing to the lungs," resulting in the patient's death and (2) that the patient was negligently left unattended in his hospital room after the operation. Before the plaintiffs can recover against any of the defendants, the Court said, it must be shown by affirmative evidence that they were either unskilful or negligent in their respective capacities and that such want of skill or care resulted in the death of the patient. "To constitute actionable negligence, there must be not only causal connection between the negligence complained of and the injury suffered, but the connection must be by a natural and unbroken sequence,—without intervening efficient causes,—so that, but for the negligence of the defendants, the injury would not have occurred. It must not only be a cause, but it must be the proximate cause." 22 R. C. L. 113.

The evidence in the record, the Court continued, pointed to no specific act of negligence on the part of any of the several defendants. Negligence, if any, must therefore be imputed from the fact that the patient was found gasping for breath, without an attendant or nurse in attendance in his room. But, said the Court, there was no evidence to show that, in the absence of the employment by the patient of a special nurse or attendant, it was incumbent on any of the defendants to see that an attendant was at his bedside at all times. Not only was there no evidence tending to prove any negligence on the part of the defendants, acting either collectively or in their several capacities, but the record contained affirmative testimony showing the proximate cause of the death to have been pulmonary embolism, such evidence being introduced at the instance of the plaintiffs. In the opinion of the Court, there must be something more than a showing that the evidence might be consistent with the plaintiff's theory of the cause of death. It must be such as to make that theory reasonably probable—not merely possible—and more probable than any other hypothesis based on such evidence. To justify a judgment for the plaintiffs, the Court said, the evidence must warrant an inference that the death resulted from the negligence of the defendants, or at least some one or more of them, rather than from some other cause. Since in the opinion of the Court the evidence in this case did not warrant such an inference, the action of the trial court in directing a verdict for the defendants did not constitute error.—*State, for Use of Kalives v. Baltimore Eye, Ear and Throat Hospital, Inc., 10 A. (2d) 612 (Md., 1940).*

Society Proceedings

COMING MEETINGS

Annual Congress on Industrial Health, Chicago, Jan. 12-13. Dr. C. M. Peterson, 535 North Dearborn St., Chicago, Secretary.

American Academy of Orthopedic Surgeons, Atlantic City, N. J., Jan. 11-15. Dr. Rexford L. Diveley, 1103 Grand Ave., Kansas City, Mo., Secretary.

American Association for the Study of Neoplastic Diseases, Washington, D. C., Dec. 18-20. Dr. Eugene R. Whitmore, 2139 Wyoming Ave. N.W., Washington, D. C., Secretary.

Society of American Bacteriologists, Baltimore, Dec. 29-31. Dr. I. L. Baldwin, Agricultural Hall, University of Wisconsin, Madison, Wis., Secretary.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1931 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Journal of Clinical Pathology, Baltimore

11:677-740 (Sept.) 1941

*Human Infection with Pasteurella Pseudotuberculosis Rodentium of Pfeiffer: Report of Case. Emma S. Moss and J. D. Battle Jr., New Orleans.—p. 677.

*Changes in Hematologic Values in Elderly Patients. W. M. Fowler, R. L. Stephens and R. B. Stump, Iowa City.—p. 700.

Pathologic Relationship of Mesenteric Adenitis, Ileitis and Appendicitis. E. B. Erskine, Jamaica, N. Y.—p. 706.

Value of Leukocyte and Differential Count in Diagnosis of Appendicitis. C. G. Smith, Philadelphia.—p. 713.

Early Carcinoma of Cervix. H. J. Schattenberg and J. Ziskind, New Orleans.—p. 719.

Malignant Thymoma: Clinical Pathologic Study of Eight Cases. C. A. Hellwig, Wichita, Kan.—p. 730.

Autopsy Permit. O. Saphir, Chicago, and C. A. Hellwig, Wichita, Kan. —p. 733.

Pasteurella Pseudotuberculosis Rodentium Infection.—Moss and Battle cite the sixth recorded case of human infection by Pasteurella pseudotuberculosis rodentium (Pfeiffer). The patient was a mulatto aged 34. The only history of contact with rabbits that could be established was that he had eaten cooked rabbit three weeks before. The strain of Past. pseudotuberculosis rodentium, isolated before death from the blood stream and after death from the blood stream, the spleen and an abdominal lymph node, was pathogenic for white mice and guinea pigs. The organism was recovered from the peritoneal exudate of the heart blood of the animals dying of the infection.

Hematologic Values in the Elderly.—According to Fowler and his colleagues, hematologic study of subjects aged 65 to 80 shows a slight reduction from the normal in the hemoglobin, erythrocyte and hematocrit values. The degree of the reduced erythrocyte count and hematocrit readings are in accord with the hemoglobin reduction, suggesting that a moderate anemia is present. Even at this age the values in women were slightly and consistently lower than those in men.

American Journal of Hygiene, Baltimore

34:39-70 Section A (Sept.) 1941. **Partial Index**

65-90 Section B 63-140 Section C 23-64 Section D

Section A

Study in Active Immunization Against Epidemic Influenza and Pneumococcic Pneumonia at Letchworth Village: I. Plan of Study. M. Siegel and R. S. Muckenfuss, New York.—p. 39.

Section B

Serologic Studies on Sugar: II. Reactions of Antiserums of Type 2 Pneumococcus and of Leuconostoc Mesenteroides with Cane and Beet Sugars and with Cane Juice. J. M. Neill, J. Y. Sugg, E. J. Hehre and Evelyn Jaffe, New York.—p. 65.

Id.: III. Reactions of Antiserums of Type 2 Pneumococcus and of Leuconostoc Mesenteroides with Sugars and Syrups of Palm, Maple and Sorghum Origins and with Sauerkraut Juice. J. M. Neill, J. Y. Sugg, E. J. Hehre and Evelyn Jaffe, New York.—p. 79.

Survey for Leptospirosis in Southern California. M. R. Greene, Los Angeles.—p. 87.

Section C

Biochemical Reactions of Genus Leishmania. H. A. Senekji and N. Zebouni, Baghdad, Iraq.—p. 67.

Evaluation of Mosquito Proofing for Malaria Control Based on One Year's Observations. R. B. Watson and Helen C. Maher, Wilson Dam, Ala.—p. 86.

Mosquito Proofing for Malaria Control from Standpoint of Construction Costs. C. C. Kiker and H. E. Breedlove, Wilson Dam, Ala.—p. 95.

Implantation of Intestinal Trichomonads (Trichomonas Hominis) in Human Vagina. R. M. Stabler, L. G. Feo and A. E. Rakoff, Philadelphia.—p. 114.

Method of Action of Atabrine on Avian Malaria Parasite, Plasmodium Cathemerium. G. H. Boyd and M. Dunn, Athens, Ga.—p. 129.

Section D

Therapeutic and Prophylactic Detoxication Anthelmintics. G. J. Martin, M. R. Thompson and N. J. Accousti, New York.—p. 23.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

46:287-446 (Sept.) 1941

Surgical Treatment of Postirradiation Necrosis. E. M. Daland, Boston. —p. 287.

Electrosurgery (Electrocoagulation, Electrodesiccation) as Adjunct to Radiation Therapy in Treatment of Cancer. G. E. Pfahler and D. A. Sampson, Philadelphia.—p. 302.

Tuberculous Tracheobronchitis: Roentgenographic Appearance. W. R. Oechsli, Olive View, Calif.—p. 312.

Hernia of Lung: Brief Review and Report of Case. A. Hartung and J. W. Grossman, Chicago.—p. 321.

Retention of Intraventricular Air Following Encephalography: Report of Two Cases of Unusual Duration. O. A. Turner, Rochester, Minn., and B. S. Brody, New Haven, Conn.—p. 324.

Roentgenologic Demonstration of Tumors of Gallbladder. J. D. Coate, Oakland, Calif.—p. 329.

Dysostosis Multiplex. M. J. Hubeny and P. J. Delano, Chicago.—p. 336.

Unusual Metastatic Bone Lesions. R. A. Rendich and A. H. Levy, Brooklyn.—p. 343.

Multiple Bony Lesions Suggesting Myeloma in Pre-Columbian Indian Aged 10 Years. G. D. Williams, St. Louis; W. A. Ritchie, Rochester, N. Y., and P. F. Titterington, St. Louis.—p. 351.

Prothrombin Formation Following Injury of Bone Marrow by Roentgen Rays. W. A. Barnes, New York.—p. 356.

Use of Iodized Oil in Roentgenography, with Animal Experimentation. T. A. Hartgraves, Phoenix, Ariz.—p. 362.

Stray Radiation Under Actual Conditions. R. B. Taft, Charleston, S. C. —p. 373.

Preliminary Studies on Dosage Measurements with Million Volt Roentgen Rays: Part I. Measurement of Quantity and Quality of Roentgen Rays in Supervoltage Range: Part II. Comparison of Tissue Doses from 200 Kilovolt and 1,000 Kilovolt Roentgen Rays. Edith H. Quimby and Elizabeth F. Focht, New York.—p. 376.

Stability of Standard Dosage-Effect Curve for Radiation. C. I. Bliss, Woods Hole, Mass., and C. Packard, New York.—p. 400.

American Journal of Surgery, New York

54:1-338 (Oct.) 1941. **Partial Index**

Anesthetic Procedures in Thoracic Surgery. S. C. Wiggin and P. E. Schultz, Boston.—p. 4.

*Postoperative Pulmonary Atelectasis. A. C. Niemeyer, Pueblo, Colo.—p. 18.

Management of Acute Empyema in Children. T. H. Lanman and C. L. Dimmler, Boston.—p. 29.

Bronchiectasis: Morbidity and Mortality of Medically Treated Patients. H. M. Riggins, New York.—p. 50.

Principles in Management of Pulmonary Abscess. R. H. Betts, Boston.—p. 82.

Recognition and Management of Mediastinitis. W. E. Burnett, Philadelphia.—p. 99.

Logical Approach to Subphrenic Abscess. H. H. Faxon, Boston.—p. 114.

Relative Frequency and Site of Predilection of Intrathoracic Tumors. B. Blades, St. Louis.—p. 139.

Tumors of Thymic Region and Myasthenia Gravis. A. Blalock, Baltimore.—p. 149.

Intrathoracic Goiter. H. M. Clute and K. B. Lawrence, Boston.—p. 151.

Carcinoma of Lung as Surgical Problem. R. H. Overholt, Boston.—p. 161.

Bronchial Obstruction: Signs, Symptoms and Diagnosis. P. Holinger and A. H. Andrews Jr., Chicago.—p. 193.

Foreign Bodies in Lung. C. Jackson and C. L. Jackson, Philadelphia. —p. 211.

Foreign Bodies in Lung. C. Jackson, Philadelphia.—p. 211.

Recent Developments in Surgical Treatment of Pulmonary Tuberculosis. E. F. Butler, Ithaca, N. Y.—p. 215.

Emergency Treatment of Pulmonary Hemorrhage in Tuberculous Patient. D. A. Cooper, Philadelphia.—p. 224.

Temporary versus Permanent Paralysis of Diaphragm in Treatment of Tuberculosis of Lungs. G. A. P. Hurley, Montreal, Canada.—p. 228.

Recognition and Management of Bronchopleural Fistula. W. Woodruff, Saranac Lake, N. Y.—p. 236.

Recent Experience with War Wounds of Chest. J. E. H. Roberts and O. S. Tubbs, St. Albans, England.—p. 289.

Problem of Wound Sepsis in Thoracic Surgery. D. Hart, Durham, N. C. —p. 295.

Deformities of Chest and Spine Resulting from Thoracic Disease and Operation: Their Prevention. J. D. Bisgard, Omaha.—p. 317.

Pulmonary Atelectasis.—Niemeyer believes that in many localities postoperative pulmonary atelectasis is still being considered as pneumonia. Follow-up study certainly bears this out, as unrelieved atelectasis often will terminate as pneumonitis or pneumonia. The idea is too prevalent that the only true expression of this condition has, as its minimal lesion, a complete lobar collapse with its mediastinal shift, and thus the minor lobular involvements are not recognized; although they do but rarely cause death they contribute considerably to postoperative morbidity and in some instances lead to pneumonia. A sudden

elevation of temperature in the first twenty-four postoperative hours, with an exaggerated rise in pulse and respiration, should suggest atelectasis. Inhalation anesthesia given to patients with productive processes in the lungs and operation on the upper part of the abdomen should be considered as potential sources of pulmonary atelectasis, and postoperative prophylactic measures (thinning the bronchial secretions, assisting in expectoration by posturing, coughing and reexpansion of the collapsed lung by deep respirations) should be instituted immediately.

American Journal of Tropical Medicine, Baltimore

21:605-716 (Sept.) 1941

*Studies on Murine Origin of Typhus Epidemics in North China: II. Typhus Rickettsia Isolated from Mice and Mouse Fleas During Epidemic in a Household and from Body Lice in Garments of One Epidemic Case. Wei-T'Ung Liu and S. H. Zia, Peiping, China.—p. 605.

Histoplasma Capsulatum: Its Cultivation on Chorioallantoic Membrane of Developing Chick and Resultant Lesions. M. Moore, St. Louis.—p. 627.

Natural Transmission of Infection from Patients Concurrently Infected with Two Strains of Plasmodium Vivax. M. F. Boyd, S. F. Kitchen and C. B. Matthews, Tallahassee, Fla.—p. 645.

Mammalian Toxoplasma in Erythrocytes of Canaries, Ducks and Duck Embryos. Fruma Wolfson, Baltimore.—p. 653.

Occurrence of Anopheles Darlingi Root in Central America. W. H. W. Komp, Washington, D. C.—p. 659.

Use of Solidified Carbon Dioxide in Developing Pressure for Spray-Killing Adult Mosquitoes in Malaria Control. F. W. Knipe.—p. 671.

Classification of Measures of Malaria Prophylaxis and Mosquito Control. P. F. Russell.—p. 681.

Temperature of Incubation for Anophelines Infected with Plasmodium Falciparum. M. F. Boyd, Tallahassee, Fla.—p. 689.

Murine Origin of Typhus Epidemics.—Liu and Zia report that during an epidemic in a household they isolated two typhus strains from mice and mouse fleas and a third atypical murine strain from body lice of one of the patients. This isolation, they believe, justifies the view that in China, as in Mexico, the human body louse may under favorable circumstances transmit the murine rickettsia from man to man, giving rise to epidemics. In their household epidemic they found that in less than a month 8 of 9 persons living in the same house had contracted the disease. The house was heavily infested with mice, the mud floor showing many holes. There were no cats or dogs. The ninth member of the household at no time throughout the outbreak showed any signs of indisposition. All the members admitted that they harbored body lice through the greater part of the year and not infrequently noticed fleas in the clothing and bedding.

Cancer Research, Baltimore

1:595-684 (Aug.) 1941

Toxicity and Carcinogenic Activity of 2-Acetaminofluorene. R. H. Wilson, F. DeEds and A. J. Cox Jr., San Francisco.—p. 595.

Further Investigation on Transmission of Induced Tumors in Fowls. J. B. Murphy and E. Sturm, New York.—p. 609.

Extraction of Carcinogenic Fraction from Human Urine: Preliminary Report. R. Steele, F. C. Koch and P. E. Steiner, Chicago.—p. 614.

Production of Tumors by Transplantation of Normally Appearing Liver Cells from Animals Previously Injected with Methylcholanthrene. W. A. Selle, P. Brindley and J. W. Spies, Galveston, Texas.—p. 618.

Transamination in Tumors, Fetal Tissues and Regenerating Liver. P. P. Cohen and G. L. Hekhuis, New Haven, Conn.—p. 620.

Induced Resistance to Transplantable Lymphatic Leukemia in Rats. E. Sturm, New York.—p. 627.

Nonspecific Nature of Induced Resistance to Tumors. M. J. Eisen and W. H. Woglom, New York.—p. 629.

Estrogenic Effects of Adrenal Tumors of Ovariectomized Mice. W. U. Gardner, New Haven, Conn.—p. 632.

Occurrence of Estrogenic Hormone in Ovarian Cysts. Ruth M. Watts and F. L. Adair, Chicago.—p. 638.

Some Cytologic Effects of Therapeutic Radiation. L. C. Fogg and S. Warren, Boston.—p. 649.

Further Studies on Effect of X-Rays on Tumor of Known Genetic Constitution. M. C. Reinhard, S. G. Warner and H. L. Goltz, Buffalo.—p. 653.

Genetics of Melanomas in Fishes: V. Reappearance of Ancestral Micromelanophores in Offspring of Parents Lacking These Cells. M. Gordon, New York.—p. 656.

Neoplasm Studies: VIII. Cell Types in Tissue Culture of Fish Melanotic Tumors Compared with Mammalian Melanomas. C. G. Grand, M. Gordon and Gladys Cameron, New York.—p. 660.

Endocrinology, Springfield, Ill.

29:165-290 (Aug.) 1941. **Partial Index**

Does Pregnancy Suppress the Lactogenic Hormone of the Pituitary? C. W. Turner and J. Meites, Columbia, Mo.—p. 165.

Effect of Light on Sexual Maturation, Estrous Cycles and Anterior Pituitary of Rat. Virginia Mayo Fiske, Cambridge, Mass.—p. 187.

Progesterone-like Effect of Ascorbic Acid (Vitamin C) on Endometrium. S. L. Israel and D. R. Meranze, Philadelphia.—p. 210.

Relation of Changes in Testicular Structure Induced in Rabbit by Estrogenic Substance to Resistance Against Syphilis. C. N. Frazier, C. K. Hu and W. C. Ma, Peiping, China.—p. 218.

Influence of Estrone on Electric Characteristics and Motility of Uterine Muscle. E. Bozler, Columbus, Ohio.—p. 225.

Hormone Content of Pharyngeal Pituitary Gland. S. Tolins, New York, and R. A. Moore, St. Louis.—p. 228.

Chronic Toxicity Studies of Diethylstilbestrol: II. Subcutaneous Implantation of Pellets in Rats. R. C. Page, New York; H. K. Russell, White Plains, N. Y.; E. L. Schwabe, C. S. Matthews and F. E. Emery, Buffalo.—p. 230.

Influence of Hormones on Phosphatase Content of Rat Femurs: I. Effects of Adrenal Cortical Substances and Parathyroid Extract. H. L. Williams and E. M. Watson, London, Ont., Canada.—p. 250.

Id.: II. Effects of Sex Hormones, Thyroxine and Thymus Extract. H. L. Williams and E. M. Watson, London, Ont., Canada.—p. 258.

Release of Glutathione Inhibition of Melanin Formation by Estrone. F. H. J. Figge, New Haven, Conn., and E. Allen, Baltimore.—p. 262.

Reproductive Organs in Malnutrition: Effects of Chorionic Gonadotropin on Atrophic Genitalia of Underfed Male Rats. M. G. Mulinos and L. Pomerantz, New York.—p. 267.

Effects of Human Pregnancy Serum on Parturition in Rat. S. S. Rosenfeld, B. Lapan and H. Baron, New York.—p. 276.

Journal of Experimental Medicine, New York

74:297-396 (Oct.) 1941

Preparation and Physicochemical Characterization of Serum Protein Components of Complement. L. Fillemer, E. E. Ecker, J. L. Oncley and E. J. Cohn, Cleveland.—p. 297.

*Studies on Agglutinogen (Rh) in Human Blood Reacting with Anti-Rhesus Serums and with Human Isoantibodies. K. Landsteiner and A. S. Wiener, New York.—p. 309.

Detection of "Masked" Virus (Shope Papilloma Virus) by Means of Immunization: Results of Immunization with Mixtures Containing Virus and Antibody. J. G. Kidd, New York.—p. 321.

Experimental Nephritis in Rats Induced by Injection of Antikidney Serum: V. Chronic Nephritis of Insidious Development Following Apparent Recovery from Acute Nephrotoxic Nephritis. J. E. Smadel and H. F. Swift, New York.—p. 345.

Quantitative Chemical Studies on Complement or Alexin: III. Uptake of Complement Nitrogen Under Varying Experimental Conditions. M. Heidelberger, M. Rocha e Silva and M. Mayer, New York.—p. 359.

Influence of Character of Antibody on Velocity of Flocculation. W. C. Boyd, Boston.—p. 369.

Experimental Gonococcic Infection of Chick Embryo. F. Bang, Nashville, Tenn.—p. 387.

Agglutinogen in Human Blood.—Landsteiner and Wiener describe a test for determing the presence or absence of the new agglutinogen (Rh), inherited as a simple mendelian dominant, in typing patients and prospective donors. The method for determining suitable serums consists in making serial dilutions by halves and testing with known negative and positive blood. Serums which show in three (or more) successive dilutions negative reactions with the former and positive ones with the latter blood are usable. Actual tests can be carried out by selecting a dilution of serum, e. g. 1:10, which gives no reactions with negative but definite reactions with positive blood, those serums that contain appreciable amounts of anti A or anti B agglutinins having been previously absorbed with small quantities of A and B blood. The blood to be tested should be fresh. An alternative method is to absorb the serums diluted, e. g. 1:4, with a quantity of blood (using A or B cells if indicated) sufficient to remove the reaction with Rh negative blood. Two drops of the test fluid are mixed with 1 drop of 2 per cent (in terms of blood sediment) freshly prepared washed blood suspension in a narrow tube and allowed to stand at room temperature. Readings are taken, after sedimentation has occurred, by direct lens inspection of the bases of the tubes. Negatively reacting blood shows a circular deposit with a smooth edge, and positive blood has a wrinkled sediment with a serrated border or shows a granular deposit. A further microscopic examination in two hours, the sediment again being inspected after the tubes are gently shaken, will reveal negative blood samples to be homogeneous, and the positive ones will show various degrees of agglutination, frequently visible to the naked eye. The blood of 448 white persons was examined with

guinea pig and/or human serums; the blood of 379 was positive and that of the others negative, that is, 84.6 per cent were Rh + and 15.4 per cent were Rh —. The blood of only 9 of 133 Negroes gave clearly negative reactions. This suggests the possibility of a racial difference in Rh distribution.

Journal of Immunology, Baltimore

41:259-374 (July) 1941

Acquired Adaptation to Anesthetic Effect of Steroid Hormones. H. Selye, Montreal, Canada.—p. 259.

Neutralization of Vaccine Virus by Immune Serum: Titration by Means of Intracerebral Inoculation of Mice. L. H. Bronson and R. F. Parker, Cleveland.—p. 269.

Extension of Separation of Types Among Pneumococci: Description of Seventeen Types in Addition to Types 1 to 32 (Cooper). Annabel W. Walter, Virginia H. Guevin, Marian W. Beattie, Hannah Y. Cotler and Helen B. Bucca, New York.—p. 279.

Studies of Passive Autosensitization and Desensitization: Two New Phenomena. O. Swineford Jr. and W. R. Mason Jr., Charlottesville, Va.—p. 295.

Absolute Rate of Phage-Antiphage Reaction. A. D. Hershey, St. Louis. —p. 299.

Optical Study of Reaction Between Transferred Monolayers of Lancefield's M Substance and Various Antiserums. J. B. Bateman, H. E. Calkins and L. A. Chambers, Philadelphia.—p. 321.

Comparison of Antibody Production by Rabbits Following Injection of Pneumococcus Vaccines Heated at 60 C. or Autoclaved. E. G. Stillman, New York.—p. 343.

Electrophoretic Examination of Digested Serums. J. van der Scheer, R. W. G. Wyckoff and F. H. Clarke, Pearl River, N. Y.—p. 349.

Leukopenic Action of Different Micro-Organisms and Antileukopenic Immunity. L. Olitzki, S. Avinery and J. Bendersky, Jerusalem, Palestine.—p. 361.

41:375-470 (Aug.) 1941

Immunochemistry of Allergens: I. Anaphylactogenic Properties of Proteic Component of Cottonseed. E. J. Coulson, Washington, D. C.; J. R. Spies, College Park, Md., and H. Stevens, Washington, D. C.—p. 375.

Strain Specificity of Complement Fixation with Serums of Mice Immune to Virus of Influenza A and Swine Influenza. M. D. Eaton, Berkeley, Calif.—p. 383.

Antigenicity of Chick Embryo. J. van der Scheer, R. W. G. Wyckoff and E. Bohnel, Pearl River, N. Y.—p. 391.

Isoantigenic Properties of Alcoholic Extracts of Brain. J. H. Lewis, Chicago.—p. 397.

Studies on Release of Histamine from Blood Cells of Rabbit by Addition of Horse Serum or Egg Albumin in Vitro. B. Rose and J. S. L. Browne, Montreal, Canada.—p. 403.

Effect of Histaminase Pretreatment on Histamine Shock in Guinea Pigs. B. Rose and J. S. L. Browne, Montreal, Canada.—p. 409.

Quantitative Studies on Group Specific Substances in Human Blood and Saliva: I. Group Specific Substance B. A. S. Wiener and I. Kosofsky, New York.—p. 413.

Immunity in Scarlet Fever: Dick Reaction, Circulating Antitoxin and Immunizing Dose. Betty S. Kolchin and I. F. Klein, with technical assistance of Irena Feig, Janet Waldman and Gertrude Cohen, New York.—p. 429.

Chemotherapeutic and Immunotherapeutic Testing of Eberthella Typhosa in Developing Chick Embryo. A. J. Weil and L. S. Gall, Pearl River, N. Y.—p. 445.

Passive Immunization Against Experimental Infection of Mice with Influenza A Virus: Comparative Effect of Immune Serum Administered Intranasally and Intra-Abdominally. R. M. Taylor, New York. —p. 453.

Military Surgeon, Washington, D. C.

89:609-736 (Oct.) 1941. **Partial Index**

Medicine and Public Health in Our Program of National Defense. F. F. Russell.—p. 609.

Meditations on a March Through the Deep South. D. E. Penhallow. —p. 616.

Medical Conditions in West Indies. G. M. Saunders.—p. 621.

Psychosomatic Medicine in Military Practice. F. P. Pignataro.—p. 632.

Quick Frozen Foods—Fish. H. E. Hess.—p. 638.

Treatment of Diseases of Salivary Glands and Ducts. V. G. Walker. —p. 656.

*United States Draftees' Dental Defects of 1863, 1864, 1918 and 1940-1941. B. R. East.—p. 661.

Recommendation for Use of Consolidated Graphic Progress Chart Toward the More Efficient Management of Gonorrhea in the Male. J. P. Pappas.—p. 664.

Treatment of Tetanus. M. L. Zee, A. F. Stein, M. F. Zeldes and M. L. Blatt.—p. 685.

Dental Defects in Draftees.—East studied the data of physical examinations of draftees of two wars and the present national emergency. Defective and deficient teeth caused the rejection of 24.2 men per thousand in 1918. The rejection rates ranged from 102.85 per thousand for Vermont to 2.9 for Arkansas. Differences in criteria probably do not explain the entire variation in rates. Dental defects defined as "loss of teeth" also played an important part in physical unfitness among the men drafted for the Northern army in the war between the states. The 1863 and 1864 draft data (Lewis, 1865) reveal that the highest rejection rates for dental defects then as in 1918 were in men from the New England states. The differences in rejection rates for the different areas in 1863 and 1864 and in 1918 correspond closely to rates of carious permanent teeth among the school children of twenty-six states examined in 1933 and 1934. The preliminary report from one hundred and twenty New York Selective Service boards shows that "missing teeth" were the most frequent cause for rejection. There is considerable evidence that the dental health of the population, as measured by physical fitness for military service, is growing worse instead of better despite a per capita increase in dental colleges and dentists and the recent extensive federal, state, city and private public dental health programs. Evidently the methods advocated and used for the last eighty years have not been sufficient to cope with dental diseases, particularly tooth decay. Since tooth formation is influenced during prenatal life, infancy and early childhood all agencies and professions concerned with maternal and child health have responsibilities and opportunities in research, education and practice in the field of dental health.

Minnesota Medicine, St. Paul

24:709-804 (Sept.) 1941

Encephalitis. C. M. Eklund, Minneapolis.—p. 723.

New Developments in Diagnosis and Treatment of Brucellosis (Undulant Fever). W. M. Simpson, Dayton, Ohio.—p. 725.

Present Day Treatment of Compound Fractures, with Analysis of Twenty-Five Consecutive Cases. T. M. Joyce, Portland, Ore.—p. 738.

Primary Treatment of Wounds. S. L. Koch, Chicago.—p. 747.

Human Blood Serum: Its Preparation and Use. R. B. Tudor and Ursula Janezich, Hibbing.—p. 749.

Psychosomatic Medicine. L. R. Gowan, Duluth.—p. 753.

Section of Anal Sphincter. H. E. Hullsiek, St. Paul.—p. 756.

Missouri State Medical Assn. Journal, St. Louis

38:303-330 (Sept.) 1941

Fetal Maternal Interdependence: Nutritional and Metabolic Factors. M. Bodansky, Galveston, Texas.—p. 303.

Significance of Abnormal Vaginal Bleeding. W. M. Allen, St. Louis. —p. 309.

Local Isolation Treatment of Boils. Jean M. Stevenson, Cincinnati.—p. 313.

Diagnosis of Pernicious Anemia. M. P. Neal, Columbia.—p. 316.

Cavernous Sinus Thrombophlebitis, Use of Serum and Chemotherapy in Its Management: Report of Seven Cases. E. S. Connell and B. C. Trowbridge, Kansas City.—p. 320.

Pregnancy Complicated by Carcinoma of Cervix: Report of Case. L. P. Kirtz and M. D. Pereira, St. Louis.—p. 324.

38:331-364 (Oct.) 1941

Significance of Hypertension and Renal Disease. C. L. Deming, New Haven, Conn.—p. 331.

Premature Separation of Placenta. S. A. Cosgrove and D. F. Conway, Jersey City, N. J.—p. 334.

*Painful Arm and Shoulder, with Especial Reference to Problem of Scalenus Neurocirculatory Compression. R. G. Spurling and E. G. Grantham, Louisville, Ky.—p. 340.

Cardinal Principles of Sulfonamide Therapy. H. F. Flippin, Philadelphia.—p. 344.

Regional Enteritis. L. S. Fallis, Detroit.—p. 348.

Progress in Anesthetic Department of Kansas City General Hospital in 1940. W. C. Schaerrer and E. H. Grandstaff, Kansas City.—p. 352.

Neurocirculatory Compression.—According to Spurling and Grantham, about 75 per cent of all patients with painful arms and shoulders (primary scalenus neurocirculatory compression) seen in the acute stage of the disorder have been improved or become asymptomatic after conservative treatment by keeping the arm up above the shoulder level with the hand behind the head as much as possible during waking hours and, if feasible, sleeping with the arm thus elevated. Tying the wrist to the head of the bed is a good way to maintain the posture during sleep. The local application of heat (the infrared lamp) over the scalenus muscle and the shoulder is also advised. Diathermy may be used if convenient. Operation is advised only for patients who fail to improve with reasonable conservative treatment.

Nebraska State Medical Journal, Lincoln

26:271-306 (Aug.) 1941

Office Gynecology. J. L. Baer, Chicago.—p. 271.
Taking the "X" Out of X-Ray Treatment. J. T. Murphy, Toledo, Ohio. —p. 278.
Blood Dyscrasias in Newborn. J. C. Moore, Omaha.—p. 282.
First Aid and Transportation of Compound Fractures. J. E. M. Thomson, Lincoln.—p. 285.
Clinical Observations on Hypertension and Diabetes Mellitus. H. H. Zinneman, Lincoln.—p. 288.
Choice of Operation for Duodenal and Gastric Ulcer. H. H. Davis, Omaha.—p. 295.
Foreign Bodies of Ear, Nose and Throat. S. Z. Faier, Omaha.—p. 298.

26:307-342 (Sept.) 1941

Study of a Patient. G. Herrmann, Galveston, Texas.—p. 307.
Problems of State Health Department. A. L. Miller, Lincoln.—p. 313.
Operation of "Blood Bank." B. C. Russum, Omaha.—p. 318.
Nutrition of Surgical Patients. J. D. Bisgard, Omaha.—p. 323.
Whooping Cough—Prevention and Treatment. F. Clarke, Omaha.—p. 328.
Earache. H. E. Kully, Omaha.—p. 330.
Epidemic Encephalitis. R. S. Muckenfuss, New York.—p. 333.

26:343-378 (Oct.) 1941

Reducing Surgical Risk in Patients with Renal Disease. H. M. Odel, Rochester, Minn.—p. 343.
The Aging Population, Its Influence on Cardiovascular Disease and a Consideration of Possible Prophylactic Measures. F. W. Nichaus, Omaha.—p. 348.
Common Mistakes in Medicine: Review of 500 Cases. F. J. Bean, Omaha.—p. 352.
Management of Some Minor Surgical Lesions of Fingers and Toes. C. W. McLaughlin Jr., Omaha.—p. 356.
Acute Appendicitis: Study of 1,006 Consecutive Cases. F. C. Hill and A. C. Fellman, Omaha.—p. 359.

New England Journal of Medicine, Boston

225:433-474 (Sept. 18) 1941

Is There a Common Denominator in Scleroderma, Dermatomyositis, Disseminated Lupus Erythematosus, the Libman-Sacks Syndrome and Polyarteritis Nodosa? B. M. Banks, Boston.—p. 433.
*Tuberculosis of Sternum. J. D. Wassersug, Middleboro, Mass.—p. 445.
Toxic Effects in Man of Overdosage with Sodium Sulfapyridine: Report of Case. F. B. Cutts and R. O. Bowman, Providence, R. I.—p. 448.
Gumma of Frontal Bone: Report of Case. N. P. Cosco, Middletown, N. Y.—p. 450.
Neurology. J. B. Ayer, Boston.—p. 452.

Tuberculosis of Sternum.—Wassersug discusses a series of 12 patients with tuberculosis of the sternum encountered among 1,134 with tuberculosis of bones and joints admitted to the Lakeville State Sanatorium between 1926 and 1940. Seven of the 12 were females and 5 males, and their ages varied from 1½ to 70 years. A history of trauma was not elicited, although 5 had had definite contact with tuberculosis. Eleven patients had other tuberculous foci. Tuberculosis of the sternum must be suspected when a swelling develops on the anterior wall of the chest in a patient with other tuberculous foci. The swelling was painless in 7 and painful in 3; in 2 a discharging sinus was the chief complaint. Eventually, one or more draining sinuses develop in every patient. Sternal tuberculosis must be differentiated from tuberculosis of the wall of the chest, ribs and costal cartilages. The tuberculosis of the sternum in 3 patients became arrested spontaneously, and the sinuses healed. In the others a variety of operative procedures was performed. The end results were usually good. Two patients are still hospitalized, and 1 died while in the sanatorium. Of the 9 discharged patients, 2 have died (1 of pulmonary tuberculosis and 1 of arteriosclerosis) without recurrence of the disease in the sternum. A third patient was taken by one of his parents to China and probably died en route. The remaining 6 patients were followed from ten months to eleven years and three months. Only slight drainage persists in the 1 patient followed for ten months. None of the other patients have had any recurrence of the disease in the sternum. All sinuses have remained healed, and there has been no swelling, pain or tenderness. However, 1 of the patients has far advanced pulmonary tuberculosis, and 1 has been bedridden for three years because of a cerebral hemorrhage. With regard to the deaths, tuberculosis of the sternum could not be regarded as a cause.

New Jersey Medical Society Journal, Trenton

38:431-494 (Sept.) 1941

Treatment of Rickets with a Single Massive Dose of Vitamin D. I. J. Wolf, Paterson.—p. 436.
The Legal Responsibility of the Physician, with Special Reference to Physical Therapy. J. Rubacky, Passaic.—p. 441.
Twenty Years' Experience in Treatment of Carcinoma of Uterine Cervix and Breast with Radium. E. A. Ill, Newark.—p. 445.
Surgical Aspects of Peripheral Vascular Disease. H. Hantman, Newark. —p. 451.
Simultaneous Immunization with Combined Diphtheria-Whooping Cough Vaccine: Preliminary Report of Eighteen Month Study. H. Simon and C. V. Craster, Newark.—p. 461.
Neurogenic Factors in Peripheral Vascular Diseases. J. F. Pessel, Trenton.—p. 465.
Cancer of Rectum. H. W. Cave, New York.—p. 468.
Nasal Hemorrhage Originating in Maxillary Sinus. R. J. Vreeland, Paterson.—p. 473.
Atrophic Rhinitis. C. C. Charlton, Atlantic City.—p. 474.

New Orleans Medical and Surgical Journal

94:155-206 (Oct.) 1941

Consideration of Fascia of Broad Ligament with Respect to Certain Surgical Procedures on Pelvis. V. S. Counseller, Rochester, Minn.—p. 155.
Acute Suppurations of Mouth, Pharynx and Neck. A. C. Furstenberg, Ann Arbor, Mich.—p. 162.
Vascular Thrombosis: Observation on Use of Heparin. W. H. Gillentine, New Orleans.—p. 169.
Rational Therapy of Thrombophlebitis. A. Ochsner and M. DeBakey, New Orleans.—p. 173.
Plasma Therapy. D. N. Silverman and R. A. Katz, New Orleans.—p. 178.
Peptic Ulcers, with Special Reference to Nonsymptomatic Type and Case Reports of Rupture into Peritoneal Cavity and Intestinal Hemorrhage. W. J. Norfleet, Shreveport, La.—p. 183.
Influenzal Meningitis: Successful Treatment of Case in Infant. Margaret Strange Bryan and J. H. Musser, New Orleans.—p. 188.
Anomalous Vein Encroaching on Fifth Lumbar Root as Cause of Sciatic Pain: Case Report. I. Redler and G. C. Anderson, New Orleans.—p. 190.
Large Solitary Abscess of Kidney. H. J. Lindner and M. E. Fatter, New Orleans.—p. 192.

North Carolina Medical Journal, Winston-Salem

2:469-528 (Sept.) 1941

Developments in Medicine. F. H. Lahey, Boston.—p. 469.
Woman's Auxiliary. Mrs. P. P. McCain, Sanatorium.—p. 471.
Physical Status of Selective Service Draftees. E. D. Peasley, Raleigh. —p. 472.
*Massive Hemorrhage from Stomach: Its Diagnosis and Treatment. R. L. McMillan, Winston-Salem.—p. 476.
Treatment of Bacillary Dysentery in Infants. W. E. Keiter, Kinston.—p. 479.
Tuberculosis and National Defense. C. D. Thomas, Sanatorium.—p. 484.
Relative Bradycardia as Sign of Sulfonamide Fever. D. Cayer, Durham. —p. 486.
Manifestations of Recent Influenza Epidemic in Adults. W. deK. Wylie, Winston-Salem.—p. 489.
Influenza in Children. C. A. Street, Winston-Salem.—p. 490.
Influenzal Involvement of Upper Respiratory Tract. J. A. Harrill, Winston-Salem.—p. 491.
Neurologic and Psychiatric Aspects of Influenza. E. A. MacMillan, Winston-Salem.—p. 492.
Mixed Influenza Bacillus-Staphylococcus Meningitis Cured with Sulfonamide Therapy. R. W. Roberts, Durham.—p. 494.

Massive Gastric Hemorrhage.—McMillan states that few medical emergencies are as hazardous as gastric hemorrhage. After the shock from the loss of blood has been compensated the cause of the hemorrhage and its extent should be determined by diligent laboratory studies and a careful history. About 90 per cent of gross gastric hemorrhages result from erosion of a vessel in the base or the margin of a peptic ulcer. The author's procedure has been to keep the patient drowsy with opiates or soluble phenobarbital, giving only small amounts of cracked ice by mouth for eight to twelve hours and then 1 ounce (30 cc.) of whole milk and 1 of cream every hour. On the second day 3 ounces (90 cc.) of a thin plain gelatin solution is given three times a day along with the milk and cream. On the third day small amounts of soft boiled eggs, cereal and toast are added, and thus the diet is increased gradually. As a rule the patient's extreme anxiety is promptly allayed and he often sleeps without sedatives. Usually bleeding stops promptly and the signs and symptoms of shock disappear rapidly. One of the greatest and most frequent errors is to give blood transfusions too soon. Giving transfusions during active bleeding results in about a 20 per cent mortality. They are indicated only if the hemoglobin level falls to 30 per cent or the falling

systolic blood pressure is 90 mm. of mercury or less, when about 250 cc. of citrated blood should be given slowly by the drip method. The low blood pressure is nature's protecting mechanism against further bleeding. Routine early surgical intervention results in a mortality of about 15 per cent, although Lahey, operating only on patients more than 40 years of age, reported a 5 per cent rate. Operation on patients with ulcer is indicated only when bleeding is recurrent or when perforation, deep penetration, obstruction, evidence of a malignant growth or gastric ulcer which after three weeks of medical treatment is still 2 cm. or more in diameter exist. The author cites 12 cases of massive and 3 of moderate gastric hemorrhage. Three patients died. The 12 who survived were given a modified Meulengracht regimen early after bleeding. Of the 3 who died, 1 received repeated small transfusions and no food, 1 died of pulmonary embolism about twelve hours after gastric resection and 1 died of postoperative peritonitis following gastric resection for a highly malignant carcinoma. None of the patients fed early died except as a result of operation performed later on.

Public Health Reports, Washington, D. C.

56:1819-1862 (Sept. 12) 1941

*Correlation Between Neutralizing Antibodies in Serum Against Influenza Viruses and Susceptibility to Influenza in Man. E. R. Rickard, F. L. Horsfall Jr., G. K. Hirst and E. H. Lennette.—p. 1819.
Diurnal Variation of Urinary Lead Excretion. S. H. Webster.—p. 1834.
Frequency of Disabling Morbidity by Cause, and Duration, Among Male and Female Industrial Workers During 1940, and by Cause Among Males During First Quarter of 1941. W. M. Gafafer.—p. 1848.

Serum Antibodies and Susceptibility to Influenza.—Rickard and his co-workers determined the neutralizing capacities of acute phase and convalescent serums against influenza A and B viruses in patients in whom the diagnosis of influenza was established by laboratory tests. The data of the study show that there are correlations between the different levels of specific neutralizing antibodies and susceptibility or resistance to the infection by the different etiologic agents of influenza. However, high antibody levels against influenza A virus do not diminish the frequency of influenza B or vice versa. Influenza of unknown cause appears to occur with somewhat increased frequency among persons who possess considerable concentrations of antibodies against both influenza A and B viruses.

56:1863-1900 (Sept. 19) 1941

*Studies on Efficacy of Complex Vaccine Against Influenza A. F. L. Horsfall Jr., E. H. Lennette, E. R. Rickard and G. K. Hirst.—p. 1863.
Present Day Methods for Controlling Aedes Aegypti Mosquitoes. J. H. LeVan.—p. 1875.
Influence of Dietary Factors on Therapeutic Activity of Sulfanilamide in Mice. S. M. Rosenthal.—p. 1880.

Complex Vaccine Against Influenza A.—Horsfall and his co-workers present evidence that a single subcutaneous injection of the complex vaccine (prepared from chick embryos inoculated with influenza A virus and the X strain of canine distemper virus) four months prior to an epidemic of influenza caused a significant reduction in the incidence of influenza A among vaccinated persons. During October 1940 7,907 volunteers of a population of 17,595 in fifteen state institutions in Florida and Alabama were given the complex vaccine. The 9,688 persons who were not vaccinated were considered as normal controls. Approximately four months after the vaccine had been given, epidemics of influenza occurred in ten institutions, with a total of 1,450 cases of influenza. In four institutions (in which volunteers received a vaccine producing about one tenth as many antibodies as the vaccines used for the other volunteers) clinical influenza occurred in 11.4 per cent of control and in 9.3 per cent of vaccinated persons, and for the other six institutions the respective figures were 8.3 and 5.6. The available evidence indicates that no level of antibodies possessed by normal persons, even though it is high, can be taken to indicate complete immunity to influenza A. The disease was less frequent in persons with high antibody levels but it did occur. The fact that the incidence of influenza of unknown cause was not significantly reduced among the vaccinated persons is additional evidence for the specific relationship between antibody levels against one influenza virus and the probability of the occurrence of infection by the homologous virus.

South Carolina Medical Assn. Journal, Florence

37:267-290 (Oct.) 1941

Study of Hookworm in South Carolina. J. C. Brabham, Union.—p. 267.
Some Considerations Regarding Constipation. J. W. Kitchin, Liberty.—p. 269.

Southern Medical Journal, Birmingham, Ala.

34:1021-1092 (Oct.) 1941

Congenital Pseudarthrosis of Tibia and Fibula: Report of Fifteen Cases. J. H. Kite, Atlanta, Ga.—p. 1021.
Fibrin Calculi. C. D. Allen, Memphis, Tenn.—p. 1032.
Trachoma Problems in Arkansas. K. W. Cosgrove, Little Rock, Ark.—p. 1037.
*Gunshot Wounds of Pregnant Uterus. T. C. Bost, Charlotte, N. C.—p. 1040.
Can the Site and Degree of Injury and Prognosis in Head Trauma Be Accurately Determined by Spinal Fluid Erythrocyte Counts? Experimental Study. J. M. Meredith, Richmond, Va.—p. 1043.
Anesthesia for Intracranial Surgery. D. H. Stubbs, Washington, D. C.—p. 1051.
Nephritis. F. G. Speidel, Louisville, Ky.—p. 1057.
Fundamentals of Treatment of Psychoneuroses. H. K. Richardson, Baltimore.—p. 1061.
Psychosoma: The Mind-Body Problem, with Special Reference to Gastrointestinal Tract. H. G. Rudner, Memphis, Tenn.—p. 1066.
*Acne Vulgaris, a Pustular Lipoidosis: Successful Treatment Based on Control of Lipid Metabolism by Low Fat Diet and Thyroid Extract. R. L. Sutton Jr., Kansas City, Mo.—p. 1071.
Laryngotracheobronchitis. V. D. Hagaman, Jackson, Miss.—p. 1082.
Girl Born Without Legs and Without Arms: Report of Case. J. L. Levy, Clarksdale, Miss.—p. 1085.

Gunshot Wounds of Pregnant Uterus.—For the thirty years since 1910 Bost finds that 9 cases of gunshot wounds of the uterus during pregnancy have been reported. He cites the tenth case. Unlike concurrent disease and trauma pregnancy apparently offers a protective mechanism against gunshot wounds. The mortality (1 of the 10 mothers died, and of the 6 viable infants 2 died) appears to be much less than the general mortality from gunshot wounds of the abdomen. Indications for opening the abdomen appear to be the same as though the pregnancy did not exist. When the abdomen is explored and no serious injury to the viscera is found except to the uterus the general opinion is that the uterus should be emptied by cesarean section if the uterine cavity has been penetrated. However, hysterectomy was done "to arrest pelvic hemorrhage" for 2 patients, 1 was operated on (the bullet was removed and the child delivered normally seventeen days later) and 1 was not operated on and labor came on and a living child was delivered normally three days later. The author's case illustrates the importance of a flat roentgenogram when there is no exit wound to determine the course of the bullet so that the proper incision can be predetermined. It shows also that a negative urinalysis does not rule out injury to the bladder.

Acne Vulgaris.—Sutton believes that an acne lesion, shallow or deep, is a foreign body reaction to oily epidermal material and suggests seborrhea. Comedo and acne vulgaris are analogous to diabetes mellitus. In diabetes, an excess of carbohydrate collects beyond the body's ability to metabolize it. In acne, an excess of lipid collects, provoking pustular foreign body reactions; acne is controllable by reducing lipid intake and enhancing lipid metabolism, if necessary, by giving thyroid. Acne vulgaris in the author's opinion is a pustular lipidosis. A carefully designed low fat diet, under supervision, is effective in diminishing seborrhea. No ill effects in more than 400 patients with acne have followed such diets. The patients had definite relief from fatigability, constipation and mental depression, while at the same time their acne improved. The constitutional symptoms of patients with ordinary acne are due to their relative hypothyroidism. By "relative" the author means the inadequacy of the autochthonous hormone to cope with the quantity of lipid ingested. Constipation is not a cause of acne; patients with acne are constipated for the same reason that they have acne, menstrual difficulties, anemia and "that tired feeling." These are all accompaniments of hypothyroidism. Thyroid is given to all patients. Objective improvement, diminution of seborrhea, is visible in a week. Improvement is obvious in a month, and from three to six months are required for cure. The diet is tentatively broken after the skin has become normal, being enriched particularly in vitamin A, and the patient determines by experiment what his lipid tolerance is.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Journal of Children's Diseases, London

38:91-122 (July-Sept.) 1941

*Varicella and Herpes Zoster: Report of Three Illustrative Cases. R. M. Campbell.—p. 91.

Varicella and Herpes Zoster.—Campbell reports 3 cases in which the vesicular eruptions characteristic of herpes zoster and chickenpox appeared concurrently. Only 1 patient was a child, following whose illness an outbreak of chickenpox affected 13 children in a ward. Sources of infection other than the infant with coexisting herpes zoster and varicella were excluded. The author believes that the unicist theory, that the viruses of the two diseases are one and the same, is inadequate to explain the facts. The absence of regular cross immunity is a serious objection to such a close interrelationship of the viruses of herpes zoster and chickenpox.

British Journal of Radiology, London

14:275-306 (Sept.) 1941

Pyelorenal Backflow. E. R. Williams.—p. 275.
Radium Distribution Factor in Carcinoma Cervix Therapy, with Comparison of Technics. B. Sandler.—p. 284.
Lymphogranuloma Inguinale Producing Rectal Stricture: Case. R. I. Lewis.—p. 298.
Homogeneity of Dosage Distribution in Radiation Teletherapy. H. T. Flint and C. W. Wilson.—p. 300.
Research into Physical Factors Concerned in Indirect Radiography: Paper V. Effect on Luminous Output of Variation of Crystal Size of Fluorescing Material. B. Stanford.—p. 304.

British Medical Journal, London

2:393-428 (Sept. 20) 1941

Operability of Carcinoma of Rectum. J. C. Goligher.—p. 393.
Parasitology of Scabies. P. A. Buxton.—p. 397.
*Treatment of Scabies: Use of Sulfur Lather Tablets. Dorothy L. Carter.—p. 401.
*Scabies Treated by Benzyl Benzoate Emulsion. I. F. Mackenzie.—p. 403.
*Transmission of Scabies. K. Mellanby.—p. 405.

Sulfur Lather Tablets for Scabies.—Carter adds a method to the list of sarcopticides which she believes has certain advantages over other methods, especially in children. The principle of the treatment was first described in America (Nolan, 1937), but instead of using sulfur soap as Nolan did the author used a tablet in which the amount of sulfur suggested as adequate, 0.72 Gm., was incorporated with a lathering agent. The lathering method is effective, especially in children, and the chance of dermatitis seems to be negligible in that the patient is given three tablets and treatment can be carried out at home without the fear of overtreatment by the zealous minded. Scrubbing was not necessary for children, in whom a hot bath and a rub-down with a flannel cloth was sufficient. None of the children complained of pain after lathering, even those with denuded or septic areas. However, the author concludes that the basic problem lies in preventing the rapid spread of the disease, and this means control and treatment of contacts and the reservoir of untreated persons. This involves inspection and treatment of every member of a family in which scabies has occurred, and also the immediate disinfestation of all their clothing and bedding. If this was applied only to school children the plan would be effective in reducing materially the incidence of scabies.

Benzyl Benzoate Emulsion for Scabies.—Mackenzie believes that the increase in scabies presents a problem of considerable complexity and of no little importance to the maintenance of the sustained national effort demanded at the present time. In a group of patients recently treated by him there was a relatively high proportion of affected families with no history of previous infection. He discusses the results obtained with a benzyl benzoate emulsion ("proscabin," Bayer). After a hot bath, the body having been well lathered with soap and scrubbed, the skin was dried and proscabin was brushed on the whole surface of the body. Badly affected parts were repainted immediately after the first application had dried. The previously worn clothing was then put on. The bath and painting were repeated on the second day and clean underwear was used. Parents were advised to change and disinfect their bedclothes. Two days later the patient was examined, and when treatment had been successful it was usual to find that scratching had ceased and that the most affected areas looked dull and brown in contrast to the many bright pinkish points normally distributed in the scratched areas. The crusted or pustular areas of secondary infection were treated with a 1 per cent ammoniated mercury ointment, after the two proscabin treatments. A second and third course of two treatments may be deemed necessary for some persons. The author suggests that the handling of infested articles (books, towels and so on) by uninfected school children may be a means of carrying scabies to previously uninfested households.

Transmission of Scabies.—Mellanby describes experiments determining exactly how scabies is normally transmitted. The experiments were made on volunteers (pacifists) living under controlled institutional conditions who agreed to submit to infection and to allow the course of the disease to be followed on their persons. The volunteers used bedding and underclothing from one to seven days after they had been used by patients with scabies and beds and underclothing immediately after being vacated and removed by patients with scabies. The men were inspected daily for about a month (sometimes longer) after each experiment. Two volunteers finally became infected, and thus it was possible to test the effects of personal contact. Four uninfected volunteers slept in beds with men with scabies. In 3 instances the volunteer and the infected man slept together for seven nights, and mites were found on all 3 volunteers after eight, nine and twelve days from the start of the experiment. Scabies did not develop in the fourth volunteer, who slept under these conditions for two nights. The author believes that scabies is normally transmitted by slight personal or venereal contact. He believes that one reason for the importance attached to fomites in the transmission of scabies is that lice are known to be spread by this means. He thinks that the evidence presented is not sufficient to say that disinfestation of clothing and blankets is unnecessary in cases of scabies, though the conclusion seems probable. When reinfestation of a patient is being attributed to fomites the following possibilities should be excluded: contact with another person with scabies, incomplete cure and as certain lesions remain visible and irritant for weeks after cure reinfestation or relapse cannot be stated to have occurred unless living mites were found.

Journal of Laryngology and Otology, London

56:189-224 (June) 1941

Injuries of Ear. H. M. Jones.—p. 189.
Mucocele of Pharyngeal Ventricle, Simulating a Laryngocele. P. Reading.—p. 204.

Lancet, London

2:331-358 (Sept. 20) 1941

Enuresis Treated by Urethral Dilatation. H. P. Winsbury-White.—p. 331.
Traumatic Asphyxia. J. G. Bonnin.—p. 333.
Plump Type of Graves' Disease. W. R. Trotter and K. C. Eden.—p. 335.
Simple Method for Estimating Proteins in Plasma or Serum. W. W. Walther.—p. 337.
Renal Glycosuria with Hypoglycemia. I. Macpherson.—p. 338.
Icterus Gravis Neonatorum Cured by Splenectomy. C. Hardwick and O. Lloyd.—p. 339.

Medical Journal of Australia, Sydney

2:249-280 (Sept. 6) 1941

Effect of Exercise in Hot Atmospheres on Salt Water Balance of Human Subjects. D. H. K. Lee, R. E. Murray, W. J. Simmonds and R. G. Atherton.—p. 249.
Review of Five Hundred Consecutive Cancer Autopsies. R. A. Willis.—p. 258.
Echinoderm Injuries in Nauru. K. V. Earle.—p. 265.
Medical Pamphlet of 1845. C. Craig.—p. 266.

2:281-310 (Sept. 13) 1941

Treatment of Certain Blood Diseases and of Hematemesis by Direct Blood Transfusion. J. A. McLean.—p. 281.
Case for Transfusion of Unmodified Blood. J. Smith.—p. 286.
Selection of Method of Blood Transfusion. A. C. McEachern.—p. 290.
Appreciation on Medical Man Power of Western Australia, with Special Reference to Economy of Personnel. N. M. Cuthbert.—p. 293.

Schweizerische medizinische Wochenschrift, Basel

71:921-948 (Aug. 9) 1941. **Partial Index**

Biologic Significance of First Year of Life in Human Subjects. A. Portmann.—p. 921.
Genital Hemorrhages as Sign of Gynecologic Disorders. H. von Wattenwyl.—p. 924.
Subacute Inflammation of Pancreas During Pregnancy. J. W. Grott.—p. 930.
Possibility of Traumatic Genesis of Closure of Vena Centralis Retinae. E. Haemig.—p. 933.
*Treatment of Influenza, of Lacunar Tonsillitis and of Otitis Media with Sulfathiazole. K. Sigg.—p. 935.
Leprosy. W. Racine.—p. 936.

Sulfathiazole in Influenza, Tonsillitis and Otitis.—Sigg employed sulfathiazole in 28 cases of influenza, 8 cases of lacunar tonsillitis and 12 cases of otiis media. It is noteworthy that the patients with influenza always show critical defervescence after sulfathiazole therapy. The temperature becomes normal in forty-eight hours. The rapid fever reduction is especially evident in the patients who have had fever for some time before the administration of sulfathiazole. In tonsillitis and otitis, the action of sulfathiazole is proved not only by the behavior of the temperature but by the disappearance of the tonsillar coating and of the redness of the tympanic membrane. In lacunar tonsillitis, the fever subsides in thirty to forty-eight hours, irrespective of the stage during which the sulfathiazole treatment is begun. The tonsillar coating disappears in two to three days, that is, the patient feels subjective improvement before the objective signs have subsided. In otitis media the fever disappears from twenty-four to thirty-six hours after onset of the sulfathiazole medication. While perforation of the tympanic membrane or running of the ear occurred in none of the 12 patients with otitis media treated with sulfathiazole, the author saw 3 patients with otorrhea for whom sulfathiazole had not been used; however, these too were cured without further local treatment when sulfathiazole was given. The author recommends large initial doses. He gives 4 or 5 Gm. on the first day, 3 Gm. on the second, 2 Gm. on the third and 1 Gm. on the fourth day. If there is no reduction in temperature at the end of the first day the larger dose given on the first day must be continued. The doses can be smaller for children. In patients with influenza the medication may cause vomiting, but sulfathiazole causes no other undesirable effects.

Revista Médica Brasileira, Rio de Janeiro

11:1-84 (July) 1941. **Partial Index**

*BCG Vaccine Against Tuberculosis. R. de Carvalho Loures.—p. 43.

BCG Vaccine.—De Carvalho Loures reports his personal experience in vaccinating 651 newborn infants from 1938 up to 1940 with BCG vaccine. He concludes, that the BCG vaccine is harmless, has no contraindications and protects infants against tuberculosis and other infections. The vaccine should be given as a routine to all newborn infants, preferably during the first ten days of life. Isolating the infants after vaccination for a given time is necessary, but the impossibility of isolating them is not a contraindication for administration of the vaccine. Infants of poor families, among whom tuberculous contacts are frequent, should be vaccinated by all means. The appearance of tuberculin allergy is the indication of the use of the vaccine by the body. Prevention against tuberculosis is maintained by repeating vaccination at certain intervals.

Revista Médica de Rosario, Rosario de Santa Fe

31:619-720 (July) 1941. **Partial Index**

*Myxedema Heart. L. González Sabathie, V. S. Terán and C. Vidal.—p. 619.
Thrombosis of Pampiniform Plexus in Vaquez Disease. R. Ercole, T. C. Minnhaar and A. Fort.—p. 644.

Myxedema Heart.—González Sabathie and his collaborators observed the behavior of the heart and of the blood pressure in 15 patients with myxedema heart, which developed spontaneously in 13 and after subtotal thyroidectomy in 2. All patients but 1, a boy, were women over 40, except 4 who were over 30. The boy was 17. The heart was greatly enlarged in 12 cases and latently enlarged in 1. The enlargement was uniform for all the chambers of the heart. In some cases the transverse diameter of the heart, as determined in relation to the transverse diameter of the thorax in teleroentgenograms, was 20 cm. The maximal arterial pressure was over 160 mm. of mercury in 4 cases and between 140 and 160 mm. of mercury in 3. There was insufficiency of the heart in 4 and angina pectoris in 3. The pulsation of the contour of the heart was diminished in all cases in which roentgenoscopic observations were performed. The electrocardiogram was changed in all cases. The most typical electrocardiographic changes were flattening of the P wave, flattening or inversion of the T wave, especially in the first and second leads, and low voltage of the QRS complex in the three standard leads. Elongation of the PR interval and widening of the QRS complex were rarely observed. There were a case of auricular extrasystole and another case of complete arrhythmia. After thyroid therapy the size of the heart diminished in all cases, except when the enlargement was due to heart disease. The arterial pressure diminished in the majority of cases. The electrocardiogram gradually became normal in all cases in a given time, which varied. Favorable changes were evident after four to eight weeks. In some cases the electrical axis changed from normal to deviated or the reverse. In a case of moderate hypertension and moderate enlargement of the heart, extrasystolic arrhythmia disappeared. The electrocardiogram had a substantial favorable change. The transverse diameter diminished 1.3 cm., without reaching, however, the normal size. In a case in which the changes of the electrocardiogram were typical and the heart was of normal size the electrocardiogram became normal after thyroid therapy and the transverse diameter of the heart diminished 1.5 cm. The size of the heart did not change in a case of severe hypertension and heart disease of arterial origin. In a case of angina pectoris the anginal symptoms disappeared during thyroid therapy and reappeared because of insufficient treatment. In 2 cases of typical insufficiency of myxedema heart the electrocardiograms became normal and the symptoms rapidly improved after the administration of thyroid therapy. In 1 there were anginal symptoms for the first time in the course of the treatment. Treatment was discontinued for some time and then was reestablished with tolerable doses. The symptoms of congestive cardiac insufficiency in 2 cases in which thyroidectomy had been done were controlled by thyroid therapy.

Münchener medizinische Wochenschrift, Munich

88:641-668 (June 6) 1941

Use of Bacteriophages in Combating Dysentery. G. Seiffert.—p. 641.
*Statistics on Kruse-Sonne (E) Dysentery: The Infectious Intestinal Disease Most Prevalent in Germany. K. Roelcke and Maria Neuberger.—p. 643.
Surgical Treatment of Visual Disturbances in Cerebral Tumors. W. Sorgo.—p. 646.
Population and Health Policies in Germany. J. Krug.—p. 651.
Question of Expelling Mucous Secretion from Bronchi. H. Weber.—p. 656.

Statistics on Kruse-Sonne (E) Dysentery.—Roelcke and Neuberger maintain that Kruse-Sonne dysentery has shown a considerable increase in Germany during the last ten years, and they present statistical data of the Heidelberg medical laboratory to prove it. This condition is not sufficiently recognized, in spite of its increase, because it may present the aspects not only of dysentery but of enteritis. They present a table which lists 825 cases in which a pathogenic intestinal micro-organism was detected at the Heidelberg laboratory between 1932 and 1940. It is noteworthy that during these nine years the Shiga-Kruse bacillus was never detected, whereas the cases of Kruse-Sonne dysentery amounted to 462, or 56 per cent, of the total number. If one looks at the figures for the different years, a rising tendency becomes apparent. Kruse-Sonne dysentery occurs not only in individual patients but in groups, and if the ages of the patients are considered it becomes apparent that the disease is most frequent in children under 10 years of age. A curve indicating the seasonal distribution discloses the highest incidence in June, July, August and September. The regional distributions suggests that the geologic character of the land may play a part. Investigating the figures on intestinal infections for all of Germany, the authors found that the incidence of Kruse-Sonne dysentery was only slightly surpassed by that of paratyphoid. Whereas cases of paratyphoid B amounted to 31.1 per cent of the total intestinal infections, cases of Kruse-Sonne dysentery amounted to 30.8 per cent. The Kruse-Sonne bacillus accounts for over 85 per cent of all cases of dysentery in Germany; the Shiga-Kruse bacillus occurs rarely. It is probable

that the incidence of Kruse-Sonne dysentery is much greater than the aforementioned figures indicate, because the clinical picture produced by this bacillus is often so mild that medical aid is not requested or that bacterial examination is dispensed with.

Archiv für Japanische Chirurgie, Kyoto

18:511-612 (May) 1941. **Partial Index**

*Results of Intrabronchial Injection of Moljodol in Chronic Empyema Patients. K. Yokota.—p. 557.

*Rosenstein's Mesenteric Pressure Pain Symptom and Its Surgical Significance. M. Matsuda.—p. 574.

Bronchial Picture in Chronic Empyema.—Yokota reports the results of his study of bronchograms of 15 patients with chronic empyema and of fistulograms in 2 children. The contrast medium employed was 40 per cent moljodol (an iodized oil) injected by the supraglottic method; in children with bronchial fistula the injection was accomplished by means of a syringe and a catheter. The three factors responsible for bronchial changes in chronic pyothorax were found to be (1) pressure on the affected side from accumulated pus, (2) scarred fibrotic changes of the alveolar parenchyma on the pleural induration and (3) contraction as a result of the pulmonary tuberculosis demonstrable in practically all cases of chronic pleural suppuration. In cases of partially involved chronic pyothorax the typical changes of the bronchi were seen in the middle and upper lobes of the right lung and the lower lobe of the left lung but never in the upper lobes on the two sides simultaneously. In total pyothorax, on the other hand, all lobes may be involved. In 4 instances the presence of a tuberculous cavity was demonstrated. Three of 4 patients in whom tuberculous bronchial changes were demonstrable in all lobes of the lungs died after extrapleural thoracoplasty, while 2 patients with no evidence of tuberculous changes of the bronchi went on to complete recovery. From these observations the author concludes that the prognosis of thoracoplasty can be predicted from the character of bronchograms. Fistula was demonstrated and the bronchi leading to the fistula showed remarkable changes, in 7 cases. These data were obtained by means of fistulography, which is also a valuable diagnostic aid in the surgery of diseases of the chest.

Rosenstein Syndrome and Movable Cecum.—Rosenstein (1920) proposed that the syndrome of mesenteric pressure pain is a simple differential diagnostic sign of appendicitis, and Torikata (1924) concluded that the Rosenstein-Inversus syndrome, which is the disappearance of ileocecal pressure pain present in the recumbent position on lying on the left side, is the pathognomonic sign of movable cecum (cecum mobile). The Rosenstein syndrome is due essentially to the pain arising from the elevation of the lateral peritoneal membrane from the retroperitoneal tissue; the presence of this syndrome in the absence of any signs of acute inflammation of the peritoneal cavity is diagnostic of movable cecum. All signs of mobile cecum may be removed by simple resection of the vermiform appendix. In all cases of movable cecum with a positive Rosenstein syndrome, the pericolic membranous tissue must be resected and the cecum must be fixed to the ascending colon; failure to carry out this procedure leads to chronic persistence of the syndrome.

Kekkaku, Tokyo

19:303-418 (May) 1941

*Experimental, Histopathologic and Clinical Studies on the Therapeutic Effect of Thymophogen on Tuberculosis. K. Ikeda.—p. 303.

Thymophogen in Tuberculosis.—Ikeda reports his observations on the therapeutic effect of sodium mono-4-chlorothymylphosphate (thymophogen) in tuberculosis. This drug was first used in 1930, when Ayukawa tested its bactericidal properties on Mycobacterium tuberculosis and found it to be efficacious because of the liberation of tetrachlorothymol in the tissue resulting from the hydrolytic action of the phosphoric acid enzymes. In animal experiments, using rabbits, the author injected 0.2 mg. of tubercle bacilli suspended in 1 cc. of saline solution either intravenously or directly into the parenchyma of the lung, simultaneously administering the test drug in doses varying from 0.04 to 0.4 Gm. as a 4 per cent solution. The injections were made every other day for a total of one hundred and sixty days. On the basis of both clinical and pathologic observations the author noted that the animals receiving injections of the drug, as compared with the control animals, gave evidence of satisfactory therapeutic effects of the drug on the disease. In contrast to the control group, the treated animals showed a greater degree of fibrosis and calcification of the tubercles in the lungs. Furthermore, the author tested the phagocytic activities of the reticuloendothelial cells, with special reference to the alveolar macrophages (called pneumocytes by Shimomura) and found that these protective elements of the body were more active in the animals with experimental tuberculosis under treatment with the test drug than they were in the control animals. As a final phase of investigation, the effect of the test drug in cases of human tuberculosis was studied. For this purpose, the author selected 30 tuberculous patients with active disease and gave intravenous injections of the drug in doses varying from 0.2 to 1 Gm. at two day intervals, for a total number varying from thirty to ninety-eight injections. Of the total number of patients so treated, 14 showed definite signs of improvement due to the drug while the remainder appeared to be unaffected.

Taiwan Igakkai Zassi, Taihoku, Formosa

40:1009-1180 (June) 1941. **Partial Index**

*Leprosy in Canton, China: A Survey. S. Rai.—p. 1133.

Leprosy in Canton.—While he was serving on the quarantine service, Rai made a survey of leprosy among the inhabitants of Canton. It has been estimated that nearly one million lepers, or approximately one third of the total number of lepers in the world, are found in China, where they are especially numerous in the province of Canton. Of the 52,000 inhabitants who came to the attention of the author, 84 (1.6 per cent) were found to show various symptoms of leprosy. Males predominated (55) over females (29); the oldest patient was 71 years and the youngest 5 years, the occurrence of the disease being most frequent in patients of the ages between 11 and 40. Of the commonly recognized clinical types, the distribution was as follows: anesthetic type 50 cases, nodular type 22 cases and macular type 12 cases. The incidence of alopecia leprosa was extremely low. There is no specific remedy in the ancient Chinese pharmacopeia. The author investigated the available therapeutic management of leprosy among the local practitioners of medicine and reports that in this field competent physicians are extremely rare. He expresses the opinion that a more adequate control and treatment of the disease is urgent if the number of lepers is to be reduced to the minimal level. He advocates institutional care of all patients with active leprosy.

Med. v. d. Dienst. d. Volksgez. in Nederl.-Indië Batavia

30:1-110 (No. ½) 1941. **Partial Index**

*A Live Plague Vaccine and the Results. L. Otten.—p. 61.

Live Plague Vaccine.—Otten, of the Pasteur Institute at Bandoeng, Java, who has studied plague immunization for more than ten years, says that opinions are divided about the potency of dead plague vaccines. After he had become convinced that it is impossible to stimulate satisfactory immunity in rats and guinea pigs by means of killed bacilli he decided to use a live vaccine. His investigations in this direction were hastened by the chance discovery of a strain that had lost its virulence completely. The favorable results obtained with a live, avirulent variant of the plague strain "Tjiwidej" in the immunization of rats and guinea pigs induced him to investigate to what extent these might also be obtained in man. He demonstrates that, according to the technic he used, the finding of avirulent variants of plague strains involves no particular difficulties and that neither the production nor the application of live plague vaccine involves unpleasant surprises or dangers. During the five years from 1935 to 1939 he employed the live vaccine in a vaccination campaign which extended to more than two and a half million subjects and in which nearly ten million vaccinations or revaccinations were done without harmful consequences to the health or life of the population.

Book Notices

Infantile Paralysis: Anterior Poliomyelitis. By Philip Lewin, M.D., F.A.C.S., Associate Professor of Bone and Joint Surgery, Northwestern University Medical School, Chicago. Cloth. Price, $6. Pp. 372, with 166 illustrations by Harold Laufman, M.D., Philadelphia & London: W. B. Saunders Company, 1941.

This book is well sprinkled with illustrations. There is a large bibliography and a good cross index. It contains chapters on the history of the disease, port of entry and physical examination. The greater part of the book has to do with care of the patient. As with any book, it represents the opinions of the authors only at the moment it was written, and these are often outdated by the time publication occurs. Some weight is given to laboratory data to support the thesis that the port of entry might be considered as of gastrointestinal origin, but little space is given to clinical evidence. The latter is the only clear-cut evidence that has consistently pointed to the fact that the disease could not possibly enter by way of the nose, despite the massive amount of laboratory work done during the last thirty years to prove this thesis. It would seem that more of this type of evidence should be mentioned.

From a physician's point of view the work is a good reference textbook. Its illustrations are easy to comprehend. One might disagree with the statement that there is such a thing as "the time relationships between the systemic phase, the period of passage through the meningeal blood vessels, the lymphatics, and the choroid plexus to the cord. . . ." Perhaps the author did not mean to imply a systemic disease with spread through the meningeal vessels, and so on. One might also disagree with the statement "Bulbar paralysis is only occasionally associated with paralysis of the spinal nerves and muscles." Nor can one agree with the statement that it is much better to keep a patient at home than to send him to a hospital. One might also disagree with the conclusion that convalescent serum should be used in large amounts intravenously and in small amounts intraspinally. The bald statement that the spinal fluid is an index of progress is only roughly true. Notwithstanding these disagreements and minor errors, the book is complete and contains a valuable appendix and all the practical information that one need know about infantile paralysis. It can be recommended. The remarks on the hypothetic means of dissemination, on the epidemiology and on predisposing factors are interesting. The author is amenable to reasoning. In fact, he states that it is quite possible that treatment might be begun a little bit earlier than heretofore recommended by most orthopedic surgeons.

Operative Surgery Including Anesthesia, Pre- and Postoperative Treatment, Principles of Surgical Technic, Blood Transfusion and Abdominal Surgery. Edited by Frederic W. Bancroft, A.B., M.D., F.A.C.S., Associate Clinical Professor of Surgery, Columbia University, New York. Cloth. Price, $10. Pp. 1,102, with illustrations. New York & London: D. Appleton-Century Company, Incorporated, 1941.

The production of a new work in surgery is an event greeted with critical interest. The mere delineation of a number of operations is not ordinarily sufficient to warrant publication. In many ways this volume justifies itself because of the fresh points of view. A number of distinguished surgeons have collaborated in presenting the various phases of abdominal surgery. The book begins with a large section on anesthesia which is actually the work of six men. The advantages of such a method are obvious, since each man writes about the particular type of anesthetic or other agent in which he is most interested. The disadvantages can be overcome only by firm editorial guidance, so that contradictions and repetitions are eliminated. Furthermore, such an arrangement may permit important omissions. It is questionable whether or not the field of anesthesia is now so complex that one or two skilled and well trained physician anesthetists could not present an adequate picture. A more unified presentation would be preferable. Nowhere in the section are the fundamental principles of anesthesia outlined. The physical signs and manifestations of the various planes of anesthesia are not presented or described by any of the six contributors, so that the entire physiologic basis of anesthesiology is omitted. The division of the section into preanesthetic medication, inhalation, rectal, intravenous, spinal and regional anesthesia is artificial, since no anesthetic is complete without the first, and the latter are frequently combined.

Other sections are concerned with preoperative and postoperative care, blood transfusion, technic, surgery of the mouth, esophagus, peritoneum and the various intra-abdominal organs. Just how surgery of the mouth is related to abdominal surgery is not made clear, and with slightly less objection surgery of the esophagus may be placed in the same category.

The contributions on these subjects, however, are generally satisfactory. In fact, much excellent information may be found on every page. This volume seems to be merely the binding together of a series of monographs. Often tabular material and other minutiae are included by some of the authors which for textbook purpose could well be further digested and condensed. Apparently insufficient effort was made to confine the contributors to their particular fields. Repetitions are numerous and needless. Perhaps fewer collaborators would have prevented some of this waste. For example, while it is conceded that the Ochsner operation for suprahepatic abscess is useful, it need not be described in detail (with the same illustrations) three separate times (pp. 499, 792, 958). Another example is the repeated description of the McBurney incision. Such repetitions probably contribute much more to the cost than to the value of the book.

Surgeons can find much of interest in this book, but unification and elimination of nonessentials would render it more practical.

Die Störungen der Blutgerinnung beim Kinde mit besonderer Berücksichtigung des K-Vitamins und der Neugeborenenpathologie. Von Professor Dr. G. Fanconi, Direktor der Universitätskinderklinik Zürich. Boards. Price, 7.20 marks. Pp. 160, with 21 illustrations. Leipzig: Georg Thieme, 1941.

The recent discovery of vitamin K and the impetus it has given to a more intensive study of the problems of blood coagulation has resulted in the publication of concise monographs both in this country and abroad. This condensed presentation first reviews and summarizes the current views on blood coagulation and the methods available for the quantitative study of the various factors. The discussion of the five main factors concerned with disturbed blood coagulability is terse except for hypoprothrombinemia, which receives the major emphasis. The author attempts to evaluate the part that calcium, fibrinogen, thrombokinase (thromboplastin), heparin and prothrombin play in bleeding states in children. Fanconi frankly states that he has never seen a case of bleeding on the basis of insufficiency of fibrinogen and only 1 questionable case in which decrease in calcium may have been a factor. The role of insufficiency of thrombokinase (thromboplastin) is concerned mainly with hemophilia, and the author critically reviews the evidence for and against the current theories. The part that heparin plays in the hemostatic mechanism is next evaluated, and Quick's and Whipple's views are mainly quoted. The subject of hypoprothrombinemia, however, receives the major emphasis in the monograph and is discussed under four main subgroups: (1) hypoprothrombinemia in sprue and celiac disease, (2) hypoprothrombinemia in liver disease, (3) hypoprothrombinemia in the newborn and (4) hypoprothrombinemia of unknown etiology. In the discussion on hemorrhage during the newborn period the author introduces a philosophical conception of extrauterine adaptation or transformation destruction to explain the dissociation of a number of vital functions which occur immediately or during the first four days of life. Whether such a speculative attempt is justified with such meager facts is questioned. The author concludes the monograph with a short summary which is rather generalized, and at the end all of the 47 cases cited are listed under their respective bleeding difficulties. The discussion is well documented with current references, and case reports are freely offered with separate case analyses. The work is concise and well written but, like all monographs on this subject, because of the paucity of conclusive data, leaves many questions unanswered. The work is stimulating and should interest those concerned clinically with the problems of disturbed blood coagulation in children.

Your Personality—Introvert or Extravert? By Virginia Case. Cloth. Price, $2.50. Pp. 277. New York: Macmillan Company, 1941.

This book represents another of the seemingly endless stream of theoretical psychologic studies that are being written and published for popular consumption. Many unsuccessful or useless attempts have been made to classify people according to type, both physical and psychologic. Although it is true that the descriptive nomenclature now employed to depict certain attitudes in human personality is extremely inadequate, further attempts to describe and clarify these unfortunate classifications only leads to more confusion. The only classifications that can possibly be of any value must be based on a dynamic analysis of the total personality of the individual and not on a descriptive classification of groups. The general public is susceptible to this sort of schematic presentation, and countless pseudoscientific publications reach the shelves of our book stores and are accepted as valuable contributions to a common understanding of human personality. In addition to the general theme of classification of all peoples into two types, introverts and extroverts, the author introduces apollonian types, archetypes, chemical types, physical types (according to Kretschmer), cycloid types, gregarious types, leptosome types, moody types, flexible types, positive types, schizoid types and Dionysian types as well as types of writers and types of intellects. There is little in this book that is of any value either to the scientific psychologist or to the layman.

Textbook of Bacteriology. By Edwin O. Jordan, Ph.D., and William Burrows, Ph.D., Assistant Professor of Bacteriology, University of Chicago. Thirteenth edition. Cloth. Price, $6. Pp. 731, with 170 illustrations. Philadelphia & London: W. B. Saunders Company, 1941.

Thirty-three years ago, when the first edition of this book was reviewed, THE JOURNAL said that it was destined to occupy a large field of great usefulness. The prophecy has been fulfilled. Long ago the book came to be widely used by medical schools and their graduates and others. Bacteriology is a comparatively young and vigorous science, which is still growing, as evidenced by the numerous editions and the gradual increase in size of the book. After the original author passed away, the twelfth edition was revised by Dr. Burrows, assistant professor of bacteriology at the University of Chicago, where the book originated. Now the cumulative effects through the decades of slowly moving developmental processes indicate the desirability of completely rewriting the book. The present edition has been constructed on a somewhat different basis than its predecessors. The chapters devoted to the highly specialized applied fields, such as soil, industry and dairy bacteriology, have been eliminated and parts of them incorporated together with new material into a chapter on bacterial physiology. A separate chapter has been written on the mechanisms operative in the transmission of infection. The fundamental principles of epidemiology have been developed through consideration of the interaction of host and parasite populations. The subjects of water and milk are considered in terms of the transmission of disease. The subject of immunity is covered in two chapters, one devoted to antigens, antibodies and the chemical basis of immunologic specificity, the other to specific resistance of infectious disease discussed in terms of humeral and cellular immunity and hypersensitivity. The many chapters devoted to the various bacteria include much new material. Consideration of the filtrable viruses has been expanded to two chapters; the first is a general consideration of the properties of viruses, and the second is given over to the virus diseases of man. This edition, like previous ones, has the references to literature at the bottom of the pages rather than in one large bibliography in the back of the book. The index generally is the same, except that now there is no separate index of authors. There is less space between the type on the page than in some previous editions. The style trends more toward technical expressions, making it perhaps a little less clear and crisp. The familiar dark blue of the binding has been changed to dark brown. The price is just double that of the original edition published in 1908, although the number of pages differs by only a hundred and seventy-four. The present author and the publishers have done well indeed in carrying on the tradition of this widely used book.

Martin Luthers Umwelt, Charakter und Psychose sowie die Bedeutung dieser Faktoren für seine Entwicklung und Lehre: Eine historisch-psychiatrische Studie. Von Dr. Med. Paul J. Reiter, Chefarzt der Psychopathenanstalten in Herstedvester. II: Luthers Persönlichkeit. Seelenleben und Krankheiten. Paper. Price, 35 Danish kroner. Pp. 633, with 17 illustrations. Copenhagen: Ejnar Munksgaard, 1941.

This is a pathography but as unlike the ordinary run of pathographic description as is a newspaper health column compared with a well controlled clinical experiment. After four hundred years Luther's life and character are still subjects of lively controversy, and an analysis of his work and personality in psychopathologic terms calls not only for erudition but in perhaps larger measure for courage and tact. The author, well known in the psychiatric field, possesses all three qualifications. It takes tact to discuss from the point of view of psychosis or psychoneurosis the inner conflicts and outer struggles of an idol worshiped by hundreds of millions and to maintain an objective approach, indulging neither in condemnation nor in justification. It takes both tact and courage to argue the delicate question whether or not excessive masturbation, intemperance or a possible syphilitic infection might have influenced Luther's life and teaching. By applying a strictly clinical method of dealing with the source material, Reiter has skilfully avoided the pitfalls of partisanship and sensationalism. After extensive differential diagnostic consideration, the final verdict is offered that the great reformer suffered from a manic-depressive condition. The possibilities of a neurasthenia or a schizophrenic or epileptic psychosis are discarded. The book is a mine of psychiatric knowledge with illuminating discussions on specific syndromes and general diagnostic issues, particularly those of temper, character and total personality. The literature is ably covered, the index is neatly arranged and the beautiful illustrations are well chosen. The volume is recommended to psychiatrists as instructive and stimulating reading.

Essentials of Pharmacology and Materia Medica for Nurses. By Albert J. Gilbert M.D., Instructor of Pharmacology, Aultman School of Nursing, Canton, Ohio, and Selma Moody, R.N., Instructor in Nursing Arts, The Presbyterian Hospital of the City of Chicago. Cloth. Price, $2.25. Pp. 251, with 15 illustrations. St. Louis: C. V. Mosby Company, 1941.

This little book is a placebo: something to soothe the student nurse and entirely without stimulating properties. It is harmless to the extent that it will do no active damage but potentially harmful, for it may leave the baneful impression that it contains the subject matter of a course in pharmacology and materia medica. The course in nursing allocates about thirty hours to this subject. It is a short period, and too much should not be attempted. However, nurses have opportunity to see drugs and their effects, which should be the equivalent of added hours. Student nurses are graduates of high schools and have been accustomed to educational methods. They should be able to assimilate much more pharmacology and materia medica than is presented in this book of "essentials." The object stated is to present the facts and theories of the subject in a lucid, concise form adapted to the limited time for the course in many nursing schools. The book is clear in statement but too dogmatic, as short statements are apt to be. It would seem better pedagogy to include more information, which would save the student's time in looking for it elsewhere and leave to the judgment of the teacher the amount and range demanded.

A Practical Manual of Diseases of the Chest. By Maurice Davidson, M.A., M.D., F.R.C.P., Physician to the Brompton Hospital for Consumption and Diseases of the Chest, London. Second edition. Cloth. Price, $13.50. Pp. 575, with 200 illustrations. New York & London: Oxford University Press, 1941.

The purpose of this volume is to make available to the internist or general practitioner for the purposes of reference a textbook on diseases of the chest but not including the heart. As the author states, "The exhaustive treatise, of vast dimensions, is too unwieldy to serve the busy practitioner; the small manual, of which there are several excellent examples, can make no pretence to be a standard of reference for those whose studies call for deeper research into the literature and pathology of the conditions dealt with. The present volume represents an attempt to supply a *via media* between these two paths." This book accomplishes the purpose fairly well. Appended to the various discussions are references dealing more exhaustively

with the various subjects discussed. The salient features of the usually encountered diseases are adequately covered and beautifully illustrated by numerous roentgenograms. Since the volume is intended primarily for physicians interested in the general field of medicine, one would welcome more extensive discussion of differential diagnosis. Particular attention is devoted to treatment, stressing particularly the practical aspects. The statement that, aside from the emergency use of bleeding for venous distention and cyanosis, "we are of the opinion that an initial venesection (in the first two days) and removal of at least 10 ounces of blood in a full blooded patient with pneumonia is a good practice and one likely to lessen the tendency to cyanosis and undue respiratory embarrassment" would not find general agreement in this country. Proprietary preparations are given undue prominence. The rapidity with which our knowledge has progressed with regard to chemotherapy is shown by the fact that the section on drug treatment of pneumonia is already somewhat obsolete and incomplete.

Untersuchungen über die Trotyl-, Tetryl- und Knallquecksilber-vergiftungen bei den Arbeitern der Munitionsfabriken Finlands. Von Leo Noro. Acta medica Scandinavica. Supplementum CXX. Paper. Pp. 95, with 7 illustrations. Helsingfors: Mercators Tryckeri, 1941.

Five years ago any publication from Finland on the toxic properties of then little known complex munition chemicals would have commanded scant attention. Today, contrariwise, this publication is of immediate significance for all physicians, chemists and engineers concerned with munitions. With arsenals springing up all over the country it becomes conceivable that "tetryl poisoning" may become as close to all physicians as the omnipresent carbon monoxide poisoning. Written in the summer and fall of 1940 by a battalion physician just after the Finnish-Soviet war, this small publication (in German) presents his observations of the damaging effect on munition workers of numerous aromatic nitro compounds, chiefly trotyl and tetryl, and the substance better known in this country as mercury fulminate. In addition to his own observations from laboratory, clinical and necropsy material, the author furnishes critical literature appraisals and an extensive literature compilation. Noro's results are not in keeping with the observations of other scientific workers in Europe on other war materials that hours of labor are outstandingly important to the incidence of industrial poisoning, but instead this work attaches prime significance to individual susceptibility calling for especial care in the selection of this class of munition workers. No less, Noro repeatedly stresses the significance of the hygiene of work places as an essence of national defense. Implied throughout the book is the concept that the immediate havoc of war is not limited to those directly attacked but may include men and women far from battle fronts and bombed areas who may be engaged in the manufacture or handling of explosive substances.

Vision: A Study of Its Basis. By S. Howard Bartley. With an Historical Perspective. By Edwin G. Boring. Cloth. Price, $3.50. Pp. 350, with 78 illustrations. New York: D. Van Nostrand Company, Inc., 1941.

This work is admittedly a handbook on the subject of vision. As such it is reasonably comprehensive, although in some few instances clarity is sacrificed for the sake of brevity. The book is well documented, the references including most of the recent works that are covered in the discussion. The author approaches the entire subject in an orderly and logical fashion, considering first the fundamentals of physiologic optics with particular reference to the entoptic phenomena in relation to their effect on the neurophysiology of the perceptive units. From this the author proceeds to the topic of neurohistology of the percipient elements and finally to the neurophysiology of all the elements involved in the visual pathway. It is to the last named subject that the author devotes most of his discussion with chapters on repeated stimulation (flicker), perception of movement, adaptation phenomena, neural interaction and contour. Discussion of the optic pathways and the cortical response are followed by an excellent conclusion in which the author points out the road that has already been traveled in attempting to elucidate this subject and what remains to be done. Unfortunately the author has considered the realm of color vision to be outside the scope of this book and has therefore left out any reference to this phase of the subject.

Die Methoden der Fermentforschung. Unter Mitarbeit von Fachgenossen. Herausgegeben von Prof. Dr. Eugen Bamann und Prof. Dr. Karl Myrbäck. Lieferung 8. Paper. Price, 34.50 marks. Pp. 2589-3047, with 86 illustrations. Leipzig: Georg Thieme, 1941.

This is a continuation and the completion of the text of a large work on methods of investigating enzymes. The bibliography and index still remain to be published. The contents of the present volume begin with page 2589. The general subject matter includes: Aspartase by Jorma Erkama and A. I. Virtanen, Helsinki. Peroxydase and catalase by Karl Zeile, Göttingen. Hydrogenase by M. Stephenson and D. D. Woods, Cambridge, England. Reducing substances of unknown mechanism: the thiol disulfide system by Theodor Bersin, Marburg, ascorbic acid by Peter Holtz, Rostock, and quinone bodies by F. Lynen, Munich. Assimilation: carbon dioxide by M. Steiner, Göttingen, and Horst Engel, Münster, nitrogen by A. Virtanen and Tauno Laine, Helsinki. Antienzymes by J. B. Sumner, Ithaca, N. Y. Ferment models by Wolfgang Langenbeck, Dresden. The enzymes of the industries: brewing by Hugo Haehn, Berlin; fat by Emil Hoyer, Rostock; malt extract by S. Preiz, Breslau; baking by H. Thaler, Munich. The distribution of authorship gives an international tone to the work, clearly of a special nature but valuable to research workers in many phases of biology. Since fermentation enters into so many phases of the kinetics of life, it is practically synonymous with life itself. As a type of the detailed inclusiveness of the work, we may select the first subject, aspartase. The treatment of this includes the naming of the enzyme, its occurrence in micro-organisms, plant and animal tissues; its preparation, purification, experiments on its action, and the products formed; the substrate; specificity; isolation and determination of the reaction products; kinetics of action; obtainability; aspartase as a reagent in the estimation of aspartic acid. This work has a place in every laboratory interested in fermentation. It should be more valuable when the index and references are published.

Essays on the Applied Physiology of the Nose. By Arthur W. Proetz, A.B., M.D., Professor of Clinical Otolaryngology in the Washington University School of Medicine, St. Louis. Cloth. Price, $7. Pp. 395, with 91 illustrations. St. Louis: Annals Publishing Company, 1941.

There is no doubt that any contribution which is intended to correlate our past and present knowledge of the physiology of the nose and sinuses is a welcome addition to a trend which has brought this field to the forefront. This monograph should serve as a challenge to workers who are interested in the field of nasal physiology to produce a definitive work. The author himself has this feeling, for in his introduction he regards his own preparation for the basic field as inadequate and offers instead a "lifetime of clinical experience" and "an absorbing interest." The author draws a corollary between the progress made by the studies in nasal pathology; he felt that if he wanted to read a specialized book on nasal physiology he would have to write it himself. The book, of twenty-one chapters, gives a sketchy outline of some of the historical contributions to this special field. On the basis of what is now known regarding ciliary action and its response to various agents, Proetz also gives an applied physiology. The structure of the nose and its relationship to function, olfaction, air currents and pressure, humidification, the mucosa as a defensive structure, vascular, lymphatic and neural reactions, are all discussed in a simple terse style based on the author's own contributions and those contained in the literature. An attempt is made with a modicum of success to correlate the commonly employed types of nasal surgery and medical treatment to the basic physiology. A universal appreciation of this principle could have a far reaching effect on the general tendency for indiscriminate intranasal medical and surgical therapy. Proetz's pioneering effort presages a constructive era in rhinology.

The Conquest of Bacteria from 606 to 693. By F. Sherwood Taylor, Ph.D., M.A., B.Sc. Second edition. Cloth. Price, 6s. Pp. 144. London: Secker & Warburg, 1940

This is a record of the historical development of the measures by which the conquest of bacteria has thus far been achieved. Intended for the layman, it is to be commended for its scientific soundness and interesting style. It achieves its purpose without resort to melodramatic distortions. Since the volume was written in England by an Englishman during the present war, American contributions to modern chemotherapy have been

almost entirely ignored. But an otherwise excellent account of sulfanilamide and sulfapyridine does not even refer to the work of Perrin H. Long or of any other American investigator. The author also creates the impression that the new chemotherapy was not warmly welcomed in America because of the "elixir of sulfanilamide" incident, which was certainly not the case. With respect to sulfapyridine in pneumonia, the author expresses the pious hope (in 1940) "that the new treatment will completely establish itself within a year or so" in the United States.

Abdominal Surgery of Infancy and Childhood. By William E. Ladd, M.D., F.A.C.S., William E. Ladd Professor of Child Surgery at Harvard Medical School, Boston, and Robert E. Gross, M.D., Associate in Surgery, the Harvard Medical School. Cloth. Price, $10. Pp. 455, with 268 illustrations. Philadelphia & London: W. B. Saunders Company, 1941.

Without doubt this is a predestined classic in its field. Abdominal surgery of children has long been regarded as difficult but, in spite of remarkable results, has been retarded by an excessive operative mortality. So much satisfaction can be attained by various surgical procedures that special attention to improve operative mortality is more than warranted. In this endeavor the senior author has long been regarded as a leader. An excellent chapter on congenital pyloric stenosis is first in the book. The various infantile and congenital disorders of the alimentary tract and its auxiliaries follow. The volume concludes with a fairly complete discussion of diseases of the male and female urogenital tracts. However, malformations associated with hypospadias are not included. Many fine photographs and drawings are included which represent chiefly the experiences of the authors and the technics they use. Statistical data and frank statements concerning the actual risk and value of various operations are an important feature. Any active abdominal surgeon should be able to perform many of the procedures described, since he is likely to possess the consummate skill and speed in operating demanded by surgery in infants.

Following a Doctor's Satchel: An M.D. Talks from Fifty Years Experience. By A. O. Scarborough, M.D. Cloth. Price, $1. Pp. 96, with portrait. Grand Rapids: Wm. B. Eerdmans Publishing Co., 1941.

Unlike most books written by venerable physicians, this one is not an autobiography. The author is still practicing medicine in a small city in Texas at 80 years of age and he simply wants to pass on the wisdom he has acquired during a long career. His advice is deeply rooted in religious principles. In his short philosophic essays he writes about love of country, paternal love, love of money, pride of ancestry, love of nature, anger, fear, sympathy. He has definite ideas about some more modern problems also. The attempted inroads of politics into the practice of medicine he has found to be very undesirable. He believes that the rise of the cults today can be explained by the prophecies in the Bible, which told us that the time would come when people would not endure sound doctrines but would run off after fables. Younger people might spend an hour, which is about all it takes to read the book, with the kindly and keen octogenarian. Interest grows as the pages go by. The editor incidentally overlooked some punctuation points, capital letters and long sentences in the opening pages of the book.

The Health and Efficiency of Munition Workers. By H. M. Vernon, M.A., M.D. Cloth. Price, $2.50. Pp. 138, with 15 illustrations. New York & London: Oxford University Press, 1940.

In this small volume the author gives a brief exposition of England's experience with hours of work, work spells and rest pauses, shift systems, sickness and absenteeism, accidents and injuries, ventilation, heating and lighting of factories, and welfare and labor management in relation to munitions output in the World War and the early days of the present war. Throughout the volume the importance of maintaining the health of the munition workers is emphasized, although he gives no specific methods. He presents a good case, however, for certain limits of productive effort, at which best results in output can be obtained without impairment of health. Appearing on the threshold of our own national defense effort, this book has value to industrial physicians, but more particularly to industrial management.

Sorting Tests in Relation to Drug Therapy in Schizophrenia. By Joseph Zubin, Ph.D., Associate Research Psychologist, New York State Psychiatric Institute, New York, and Jane Thompson, Ph.D., Psychological Assistant, New York State Psychiatric Institute. Paper. Pp. 23. New York, 1941.

This small monograph was undertaken to check the findings of another author regarding a supposed relationship between patients' performances before and after insulin and metrazol shock therapy. The sorting tests were purely psychologic with a statistical analysis. The tests used were the Weigl Color Form, the Vigotsky and the BRL sorting. One hundred and fourteen patients were studied. The conclusions were that the sorting tests showed the existence of a definite relationship between patients' performances before and after shock therapy. There is a good bibliography. This publication is recommended to all psychologists.

Standard Radiographic Positions. By Nancy Davies, M.S.R., Senior Radiographer, Royal Surrey County Hospital, Guildford, and Ursel Isenburg, M.S.R., Radiographer to the Charterhouse Rheumatism Clinic, London. Cloth. Price, $2. Pp. 136, with 103 illustrations. Baltimore: William Wood & Company, 1941.

This is a little handbook on radiographic positioning, of such size as to fit into the pocket of a coat or gown and intended to serve as a ready reference book for standard radiographic positions. For details of technic of special x-ray examinations the reader is referred to more elaborate books. The illustrations are diagrammatic, only the main structures and outlines being given. In view of the needs of these war days, considerable space is devoted to positions required in the x-ray localization of foreign bodies. In perusing this work one finds little to criticize. It is not a pretentious publication, but it serves well for the purpose intended and should be valuable for radiographic technicians.

Die tägliche gynäkologische Sprechstunde. Von Dr. M. Rodecurt. Cloth. Price, 10.50 marks. Pp. 308, with 3 illustrations. Leipzig: Georg Thieme, 1941.

This book, which is devoted to office gynecology, has nineteen chapters including those dealing with infections of the genitalia, malpositions of the uterus, abnormal uterine bleeding, endocrinology, sterility, habitual abortion, pregnancy, diseases of the breasts, backache, rheumatic-neuritic-neuralgic-vegetative symptom complex, the menopause, cosmetics and sexualia. Some of the chapters are disproportionately large. The one dealing with Trichomonas occupies forty pages, or one seventh of the book, whereas only eight pages are devoted to gonorrhea. There are only three illustrations in the entire book, two of which depict Trichomonas and the third shows the Knorr instrument, which is used for applying silver nitrate to the bladder in cases of urinary incontinence. The book is well written, but it loses a great deal of its value because of the almost complete lack of illustrations, which are essential for the instruction the author attempts to give.

Necropsy: A Guide for Students of Anatomic Pathology. By Béla Halpert, M.D., Assistant Professor of Pathology and Bacteriology, Louisiana State University School of Medicine, New Orleans. Cloth. Price, $1.50. Pp. 75. St. Louis: C. V. Mosby Company, 1941.

This brief manual, written primarily for students, describes in a systematic manner the procedure for conducting a necropsy. The anatomy of the organs and their anatomic relationships with associated structures are emphasized in twenty brief sections. No discussion of any pathologic alterations of the organs is attempted. The principles for a complete necropsy record are stated in the appendix. Two sample records including anatomic and microscopic descriptions are given as examples of a representative report. The book is entirely devoid of illustrations. The medical student and intern will find this a handy book for quick reference.

Laboratory Guide in Elementary Bacteriology. By M. S. Marshall. Fabrikold. Price, $1.75. Pp. 244. Philadelphia: Blakiston Company, 1941.

This little laboratory guide is designed to serve a course in elementary bacteriology. It is, however, a little astonishing to find listed the nine volume System of Bacteriology and Topley and Wilson's two volume Principles of Bacteriology and Immunity as textbooks for an elementary course. The value of this guide can be determined only under conditions of actual use and will doubtless be found satisfactory for some courses and not for others.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

NAPOLEON'S URETHRAL STRICTURE

To the Editor:—**Some years ago in a paper on the history of Napoleon the assertion was made that the battle of Waterloo was lost because Napoleon, on account of a urethral stricture, could not pass his urine. Can you give such information as would lead to a corroboration of this statement?**

Edward J. Ill, M.D., Newark, N. J.

ANSWER.—Dr. Edmund Andrews, in a paper entitled The Diseases, Death and Autopsy of Napoleon I, published in THE JOURNAL, Dec. 21, 1895, page 1081, presented evidence on which the assertion mentioned in the query may have been based. Andrews in that article quoted Field Marshal Garnett Wolseley, who was at that time (1895) head of the British army, as stating "that at the battle of Waterloo Napoleon suffered pain in riding, and that he had a table set in the open air in a suitable position for receiving dispatches and issuing orders, where he sat for hours, much of the time asleep, leaning his head on his arms." Further in Andrews' article the following sentence appears: "All this terrific labor he accomplished while suffering under a genitourinary disease which may have partly weakened his force of will." Andrews continues: "During the Russian campaign in 1812, Napoleon began to have difficulty in expelling his urine." Andrews states that there can be but little doubt that the trouble was urethral stricture with cystitis. He says that the sequence of events seems to have been (1) an attack of gonorrhea, (2) a stricture of the urethra, (3) a cystitis, (4) nephritis with a few small calculi and (5) albuminuria, dropsy of the legs, and uremic poisoning in a moderate degree.

FAMILIAL TREMOR

To the Editor:—**A woman aged 54 has had intention tremor of the head and hands since childhood. Her father, one sister out of a family of eight, and several cousins have all had the same tremor, although to a lesser degree. The tremor becomes much worse under stress and strain and almost completely disappears when she is at rest. I have tried 1/200 grain (0.0003 Gm.) of scopolamine three times daily without success. One and one-half grains (0.1 Gm.) of phenobarbital at a time gives only slight relaxation and does not make her at all drowsy. Alcohol is the only drug which will give her complete, although temporary, relaxation. After she has drunk about half a cocktail her hand will become so steady that she can lift the glass without spilling any liquid. At other times she has to drink through a straw. Can you tell me of any other drug which will have a similar reaction?**

Elizabeth Brakeley, M.D., Montclair, N. J.

ANSWER.—There is no drug as useful as alcohol in abolishing a familial tremor. The effect, however, is temporary and this method of therapy cannot be used in a routine manner.

Not many experiments have been made on tremors of the familial kind, but there is a large literature with regard to the effect of drugs on the tremors of parkinsonism, either of the postencephalitic or of the paralysis agitans type. Ziskind (THE JOURNAL, July 3, 1937, p. 20) has shown that phenobarbital tends to increase the rigidity of chronic encephalitic parkinsonism, and Friedman's work (*Bull. Los Angeles Neurol. Soc.* **5**:120 [June] 1940) indicates that the same drug decreases the steadiness and increases the tremor. Scopolamine has long been recognized as a drug having the effect of releasing the rigidity of paralysis agitans but at the same time making the tremor worse either through the lessening of the rigidity or by some direct action on the nervous mechanism. Neither phenobarbital nor scopolamine would seem likely, therefore, to be of value in controlling a familial tremor.

The occurrence of tremors in animals deprived of vitamin E, as shown by Mackenzie (*Proc. Soc. Exper. Biol. & Med.* **44**:95 [May] 1940), has led to the use of alpha-tocopherol in patients suffering from tremors of various types. The results, so far as known, have not been beneficial, but no published series has yet appeared in the literature. This method of treatment is worthy of further trial, although the outlook for a favorable result is not good.

A more promising field lies in the use of the belladonna alkaloids. Raeff, a Bulgarian plant collector, was first to use the extract of belladonna root in 1926, and the treatment of parkinsonism by this method was intensively studied in Italy by Panegrossi (*Policlinico* [sez. prat] **42**:46 [Jan. 14] 506, [March 18] 1935). The results were uniformly good both in Italy and in Germany. The preparation used was a white wine extract of the whole belladonna root. Later American, English and Italian belladonna root was substituted for the original Bulgarian product with equally good results. The extract is not well tolerated by the aged, the arteriosclerotic or the alcoholic. Toxic symptoms in other groups of patients are rarely seen.

From the work with belladonna root in parkinsonism, it was concluded that the therapeutic effect of the belladonna alkaloids is increased when they are given in combination, as in an extract of the root. When the purified alkaloids are extracted and put together in a tablet, the results are equally good, as shown by the work of Gayle and Pettis (*Am. J. Psychiat.* **97**:1175 [March] 1941). They used a compound of atropine, scopolamine and hyoscyamine. Their report is based on the treatment of parkinsonism alone, without cases of familial tremor. The results were striking and the combined drugs were not dangerous, with ordinary precautions, to use. Riebold (*München. med. Wchnschr.* **87**:1183 [Oct. 25] 1940) reports one case of familial tremor treated with the belladonna root extract, with surprisingly good results. Either the extract or the compound tablet may be tried, therefore, in this case.

EXFOLIATIVE DERMATITIS AND ALOPECIA

To the Editor:—**A man aged 24 complains of complete loss of hair of the scalp and inability to sweat, except on the hands and face. He suffers extremely during the summer months and can hardly do any work. He had a skin condition two and one-half years ago, at which time his skin peeled off several times over a period of four months and during which he lost all his hair. (This sounds like an exfoliative dermatitis.) Following this he began having trouble with his sweat glands. Physical examination and laboratory tests are negative. Examination of the skin reveals the hands and face to be moist. The rest of the skin is extremely dry. What is this condition? Is there any treatment for it? Would a biopsy of the skin give any information?**

M.D., South Dakota.

ANSWER.—The man probably had an exfoliative dermatitis, a symptom complex which may be due to divers causes. The history is not sufficiently informative to differentiate among these several causes. The primary or type of Wilson-Brocq exfoliates with large scales over a prolonged period and is accompanied with few constitutional symptoms. The inference is that this man's exfoliative dermatitis did not last long. The secondary type of exfoliative dermatitis appears as a complication of some preexisting eruption such as psoriasis, lichen planus or pityriasis rubra pilaris, which has been too strenuously treated or which has spread, to become universal. The history does not mention anything about a preceding eruption. Pityriasis rubra of Hebra exfoliates with fine branny scales; the skin becomes pigmented and bronzed and atrophic. This is supposedly on a tuberculous basis, but some patients who have had this dermatitis are known to have developed frank leukemia. This entity too is long lasting and would not seem from the history to have been present. One of the several members of the lymphoblastoma group—leukemia, Hodgkin's disease, lymphosarcoma and granuloma fungoides—may give rise to exfoliative dermatitis as one of their nonspecific varieties of eruption. Eventually some specific sign of their presence, such as changes in the blood or type of clinical and histologic cutaneous change, makes them recognizable with certainty. This patient would not seem to be suffering from anything in the lymphoblastoma group of diseases. Exfoliative dermatitis may be on a toxic basis, as in idiosyncrasy to one of the arsphenamines.

The complete loss of scalp hair dating from the time of the exfoliative dermatitis may be assumed to have been part of the latter process. Loss of hair and nails not uncommonly occurs with exfoliative dermatitis. It is usual, however, for the hair to regrow. The history does not mention anything with regard to other hair than on the scalp. There is a possibility that the loss of hair may be an extensive alopecia areata. In this, one would expect the hair at other sites than the scalp to be affected.

Alopecia areata is a somewhat attractive diagnosis, because in it there is supposedly present a functional imbalance or disorder of the vegetative nervous system. This seems a probability here because of the excessive sweating of the palms. Excessive sweating may be due to debilitated states and toxic processes, but in such cases it is more generalized. In hyperhidrosis due to organic nervous changes, the sites may be localized but are not apt to be symmetrical as is the fact with

this patient. Excessive perspiration of the palms and soles commonly accompanies emotional tension. Sweating may be induced by direct or pharmacologic stimulation of components of the vegetative nervous system and, contrariwise, diminished or absence of sweat secretion follows their depression.

The probability exists that the patient has some functional nervous disorder. The services of a neuropsychiatrist would be valuable. The patient should be reinvestigated to rule out some source of toxemia or endocrinal change. Can anything abnormal be found with regard to the pituitary body by roentgen ray examination? If nothing definite can be found to account for the patient's troubles, certain empirical and symptomatic agents may be useful.

The patient should be withdrawn from an environment and situations which increase emotional tension. Certain drugs may be helpful, such as the use of antispasmodics. Thyroid may be cautiously tried. For excessive sweating of the palms and soles, roentgen therapy, one-fourth unit at weekly intervals for four doses, may be helpful. For the same also application of 25 per cent aluminum chloride in aqueous solution each night for ten days and then twice a week thereafter. For the anhidrosis of the body, the cautious use of pilocarpine is indicated. Keeping the patient in a cool environment is necessary to prevent discomfort and even danger. A course of ultraviolet exposures to the scalp is one indication for the alopecia. Another method is the use of direct stimulants such as the application at weekly intervals of Cutler's fluid (equal parts of chloral hydrate, phenol and tincture of iodine), but if skin irritation appears it must be promptly discontinued.

PYORRHEA ALVEOLARIS AND ARTHRITIS

To the Editor:—**At the various symposiums on infectious arthritis which I have attended during the past several years, the consensus seemed to be that pyorrhea could not be regarded as a contributing focus of infection. I have been advised that some of the leading dental schools feel that this is a mistaken assumption. They believe that this infection is picked up by the lymphatics and is thus carried through the system and that, furthermore, the more modern treatments of pyorrhea block the lymphatic drainage from affected areas and thus prevent additional absorption. Please comment on these views.**

Robert Hoffman, M.D., South Bend, Ind.

ANSWER.—It is assumed that by the term "infectious arthritis" the writer is referring to the so-called chronic nonspecific infectious or rheumatoid (atrophic) arthritis. Despite the enormous amount of work done during the past seventy-five years to prove the microbic theory of rheumatoid arthritis, the cause of this disease is still unknown. It has not yet been definitely established that the disease is due to bacteria or even to bacterial toxins, nor has the role of infected foci been accurately determined. In the United States the majority of students of the rheumatic diseases are withdrawing from their former attitude of zealous adherence to the old idea that infected foci were a frequent cause of arthritis and that the removal of such infected foci could be expected to modify notably the course of the disease. The pendulum has swung away from the theory of focal infection simply because the results from the removal of various infected foci have been in the main so disappointing. Indeed, one of the prominent former advocates of removal of infected foci recently has stated that "The time has arrived for a complete revaluation of the focal infection theory. Undoubtedly there are cases of infectious arthritis which result from focal infection. However, as far as typical rheumatoid arthritis is concerned, it would appear that chronic focal infection plays a comparatively unimportant role" (Cecil, R. L., and Angevine, D. M.: *Ann. Int. Med.* **12:** 577 [Nov.] 1938). One view is that many infected foci may be the result, not the cause, of the disease; that is, that as the result of the general debilitating effect of rheumatoid arthritis the whole body is "run down," the general resistance of various tissues is lowered and, in some tissues which have become seats of lessened resistance, focal infections may develop secondarily (Pemberton, Ralph: *J. Bone & Joint Surg.* **53:**879 [Oct.] 1935).

Infected teeth and pyorrhea are commonly found on examination of patients with rheumatoid arthritis, but they are also commonly found on examination of nonarthritic persons and of patients with forms of chronic arthritis (for example, osteoarthritis) not considered infective in nature. The majority of physicians and dentists have considered periapical infections more potent foci than gingivitis or pyorrhea. But representative of another view is the belief of J. V. Cogan that gingivitis is more important than periapical infection in cases of chronic arthritis and that such gingivitis requires special dental therapy and not just cleaning (*J. Am. Dent. A.* **26:**56 [Jan.] 1939). In contrast with this opinion is that of another student of rheumatic diseases who considered gingivitis "unimportant" and root abscesses "greatly overrated as a source of arthritis" (Monroe, R. T.: Oxford Loose-Leaf Medicine, Oxford University Press, 1939, chapter 15, pp. 367-404).

In support of the idea that gingivitis may play a role in the production of chronic arthritis can be mentioned the work of Cook and Stafne (*Dental Cosmos*, February 1939), who recovered organisms, chiefly Streptococcus viridans, from deep pyorrhea pockets of patients who had "chronic infectious arthritis" and, by injecting them intravenously into rabbits, were able to produce acute purulent arthritis in the rabbits, from the joints of which Streptococcus viridans was readily recovered. But in interpreting these results it must be noted that the articular lesions (acute purulent) in the animals did not resemble the chronic nonsuppurative lesions of the patients studied. Such experimental studies will be difficult to interpret until some characteristic bacteria or bacterial toxin can be consistently isolated from the joints or blood stream of patients who have rheumatoid arthritis or at least until the course of rheumatoid arthritis can be shown to be more significantly and more frequently modified by the removal of infected foci than has been shown to date.

The present attitude of most American rheumatologists is that arthritic patients with infected foci are certainly "less fortunate" than those without infected foci. Infected foci, including gingivitis, may well be harmful to the arthritic patient in a general way, if not in a specific way; hence their eradication is indicated, but no promises should be made that the articular disease will be notably altered thereby.

Much can be accomplished in the treatment of teeth affected with pyorrhea if the situation is recognized early before too much of the supporting tissues have become involved.

Treatment consists in thorough cleaning of the teeth, polishing of the exposed root surfaces, elimination of deep pockets and hypertrophied tissue and correction of traumatic factors. The patient should be instructed with regard to the necessity and importance of dental hygiene.

Generally speaking, if two thirds of the bony support of the single rooted anterior teeth is lost, or if the bifurcation or trifurcation of the multirooted teeth is exposed, the general practitioner should consider these teeth beyond treatment and they should be removed.

TREATMENT OF DEMENTIA PARALYTICA

To the Editor:—**Should a middle aged patient with dementia paralytica whose serologic reaction has become negative after antisyphilitic treatment but whose clinical picture shows deterioration in spite of the negative serology be continued with treatment? Opinion seems to differ. According to the Vienna school one should keep on treating and one should go by the clinical picture rather than by the serologic findings. However, it seems that according to certain American schools of thought one would stop treating a patient such as the one mentioned.**

M.D., Iowa.

ANSWER.—The answer to this question depends on whether the patient has already received fever therapy, preferably with malaria. If so, the situation is as summed up by Moore (The Modern Treatment of Syphilis, ed. 2, Springfield, Ill., Charles C. Thomas, 1941, p. 443): "Though improvement in the mental status may occur actually during the course of fever, it may also be delayed for several weeks or months. Worldwide experience has demonstrated, however, that if a change for the better does not appear within six months it probably will not occur at all. If malaria and the appropriate after-treatment have been unsuccessful in effecting any betterment during this period of time, the chance of obtaining it from any form of treatment, even including a second trial of fever therapy, is so small as to make further therapeutic efforts unjustifiable unless the family's finances are almost unlimited." This applies, however, only to malaria-treated paretics. If malaria has not already been employed for a patient such as the one described, it should be given a trial.

SULFHEMOGLOBINEMIA AND TUMOR GROWTH

To the Editor:—**I have observed some cases in which sulfhemoglobinemia has seemed to retard the growth of malignant tumors and to make such tumors more sensitive to roentgen rays. I would like to have an informed opinion on this observation; furthermore, I would appreciate any reference to the literature.**

M.D., Indiana.

ANSWER.—So far as is known there is no evidence that sulfhemoglobinemia retards the growth of tumors or renders them more sensitive to roentgen rays.